This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain articles, news items, reports, digests, and other presentations, together with editors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited.

Industrial Medicine

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The editors will exercise care in checking on the accuracy of data printed, but in all other respects articles and opinions of which expression is allowed are the opinions of their authors—the editors reserving in all cases the right to comment on the same, in the current or any subsequent issues, as they may be inclined.

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Tuberculosis Incidence in Philadelphia's Industries

— Present Status and Suggested Improvements —

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WHAT I wish to bring to you is the conception of pulmonary tuberculosis as we who work in industrial medicine find it, what we do to discover it, what type of thinking we do about it, how we care for the employee who has it and how we try to prevent its development and spread.*

For years industrial physicians have been very much alive to tuberculosis. The studies of Louis Dublin[1.a,b,c] from 1911 through 1935 have put us on our mettle. Of general industry he said: "The death rates for tuberculosis, age period for age period, are from two and one-half to nearly four times as high among the industrial workers as among the professional and agricultural groups." And of my own industry — cigar making: "The deaths from tuberculosis of the lungs were found to be one and one-half times the normal." Reading these figures, one would have cause for concern, and added to these sources of worry, we have the now legally recognized association between silicosis and pulmonary tuberculosis. Finally, there is the relationship between chest trauma and self re-infection from spilled cavity contents, or the rupture of a none too well healed lesion.

Industrial physicians have either invented or adopted a great many techniques which in themselves are not a part of an anti-tuberculosis program, but nevertheless effect that result. Pre-employment examination, periodic health examination, examination after illness or upon re-employment, all serve as processes for detecting the individual with pulmonary tuberculosis. Here is our greatest contribution to the general problem. In preparing this report I submitted a questionnaire to the Medical Directors of those Philadelphia industries whose medical services have the certificate of approval of the American College of Surgeons.[2] Chart I is prepared from answers representing medical responsibility for 236,193 employees. Without exception every physician in these industries requires a physical examination before the applicant is accepted for employment. The inevitable by-product of such wholesale examinations must be the discovery of pulmonary tuberculosis.

This chart also shows the wide divergence in industrial medical practice and gives us opportunity for constructive suggestions that will certainly improve our tuberculosis control. This organization (No. 7) requires pre-employment and periodic health examinations, including in every instance stereoscopic chest film. In addition, their own laboratory examines sputum for tubercle bacilli. On the other hand, here are two industries (No. 15 and No. 5) each with 3000 employees, who require pre-employment examination and nothing else. It is obvious that the incidence of any disease, including tuberculosis, must be better known to the medical director of the first industry, than of the other two.

At present one notes there are several industries where these periodic health examinations are not mandatory as are the pre-employment examinations. They are used only in groups with special hazards. The difficulty here lies in the fact that disease will arise in any employee regardless of hazard. And the hazard of tuberculosis is greater in the home than in the factory. Consequently, if we wish to discover our total illness incidence, we are forced to adopt routine periodic health examinations of the entire personnel. But, a majority of those reporting *have* instituted periodic health examinations. This augurs well for their eventual complete adoption and will be

* Presented at a Tuberculosis Symposium of the Section of Public Health, Preventive and Industrial Medicine, Philadelphia College of Physicians, December 5, 1938.

CHART I.

(PREPARED FROM ANSWERS REPRESENTING MEDICAL RESPONSIBILITY FOR 236,193 EMPLOYEES)

				Case Finding							Case Care		Expense Borne			
Code No.	Number Employees	No. Tuberculars Active	No. Tuberculars Inactive	Pre-employment Examination	Health Examination	Fluoroscopy	Paper Film	Standard Film	Laboratory	Mantoux	Family Phys.	Clinic	Company	Beneficial Association	Disability Insurance	Company Sanitarium
1	10,000	8	42	+	+	+	—	—	+	—	+	+	—	—	—	—
2	4,000	1	4	+	+	+	—	±	—	—	+	+	±	—	—	—
3	16,593	7	40	+	±	—	—	±	+	—	+	+	—	—	—	—
4	1,500	0	0	+	+	±	—	—	—	—	+	+	—	—	—	—
5	3,000	0	1	+	—	—	—	—	—	±	+	+	—	—	—	—
6	2,000	0	0	+	±	—	—	—	—	—	+	±	—	—	—	—
7	1,700	1	11	+	+	—	—	+	+	—	+	+	+	—	—	+
8	1,500	0	1	+	—	—	—	—	—	±	+	+	—	—	—	—
9	2,500	0	15	+	±	+	—	±	—	—	+	+	—	—	—	—
10	3,000	4	2	+	+	—	—	—	—	—	+	+	—	+	—	±
11	4,000	0	11	+	+	—	±	—	±	—	—	+	±	—	—	—
12	800	2	0	+	+	—	—	—	—	—	+	+	—	—	+	—
13	10,000	0	0	+	—	—	—	+	+	—	+	—	—	—	—	—
14	140,000	50—60	?	+	±	—	±	±	+	—	+	+	—	+	—	—
15	3,000	5	5	+	—	—	—	—	—	—	+	+	±	—	—	—
16	9,000	0	13	+	±	—	—	+	+	—	—	±	—	—	—	—
17	15,000	17	?	+	+	—	—	±	±	—	+	+	—	+	—	—
18	3,100	0	2	+	—	—	—	—	±	—	+	+	—	—	—	—
19	1,000	6	20	+	—	—	—	—	+	—	+	+	—	—	—	—
20	4,500	0	0	+	—	—	—	—	+	—	+	+	—	+	—	—
Total	236,193															

+ = Mandatory ± = Optional

our next great contribution toward tuberculosis control.

From the tuberculosis standpoint every pre-employment or routine group health examination should include a chest film. We are still far from that ideal, since only three industries take routine films. In my own experience the fluoroscope has been of great aid. In 1936 I became interested in the chest fluoroscopic technique and work of Dr. Ada Chree Reid,[3] of the Metropolitan Life Insurance Company. Since that time all pre-employment and health examinations done by me have included a fluoroscopy of the thorax. Consequently, I have a great deal more confidence in my figures on tuberculosis incidence now than previously. It is a pleasure to see that one large industry (No. 1) and one smaller one (No. 9) stand with me in this conception.

Britton[4] recently stated that in a large industrial group the tuberculosis incidence rate was 80 per 100,000. The Committee on Administrative Practice of the American Public Health Association[5] assumes that in any community or group there are five living active and arrested cases of pulmonary tuberculosis for every reported death. The following statistical table prepared by Whitney[6] therefore becomes of great importance to this discussion:

TUBERCULOSIS DEATH RATES PER 100,000 EMPLOYED MALES AGE 15 - 64 YEARS

Professional Men	26.2
Proprietors—Managers—Officials	43.2
Agricultural Workers	46.5
Clerks and Kindred Workers	65.8
Skilled Workers and Foremen	72.1
Semi-skilled Workers	102.1
Unskilled Workers	184.9

Multiplying each figure by 5, we would have 131 living tuberculous professional men in every 100,000 as compared to 910 tuberculous unskilled workers per 100,000!

Now, to return to our own Philadelphia industries (Chart I). We must omit these two (No. 14

and No. 17) whose figures are not available, but for the 18 others we find 201 known tuberculous among 81,193 employees, or an incidence rate of 251 per 100,000. This stands midway between the expectancy for officials and that for clerks in Whitney's table. Certainly it is better than the expectancy for any of the classifications into which these 80,000 employees actually fall.

It is even better than the Philadelphia incidence rate for pulmonary tuberculosis which we calculate from Lowell's[7] morbidity data. He cites a pulmonary tuberculosis mortality of 58.3 per 100,000 of Philadelphia's population for 1937. This would warrant an expected morbidity of 291 tuberculous per 100,000 individuals.

For all the dusty industries, including tobacco, the American and foreign literature[8] was rife with statements purporting to show a marked relationship between dust and pulmonary tuberculosis until the work of H. R. M. Landis[9] for the entire group, and T. Grier Miller[10] for the tobacco industry, began to lift the cloud of suspicion. Landis quoted Beattie, saying: "The dust is not nearly so dangerous to life as the tubercle bacillus. If you get rid of the bacillus of tuberculosis, the dust is not so bad." Miller, in his study of 400 cigar factory workers, found no active tuberculosis and only six with chronic fibroid apical infiltration. In this connection I wish to report the results of our two mass health examinations. The first surveyed 4145 cigar factory employees, of whom three were found to have active, and eight healed, tuberculosis. The active patients were immediately referred to their own physicians with their x-ray findings, and treatment instituted. Three years passed and we again examined 4017 employees. Four of our known arrested cases were still with us in good health. Only *one* new active case was discovered. This means a present incidence rate of 125 tuberculous per 100,000 employees.

This figure and those of the reporting Philadelphia group seem to refute Dublin's statements which I quoted above. His statistics are not opinions; they are cold facts culled from the death records of a large insurance company. But neither are our findings merely wishful thinking. A suggested explanation of the discrepancy is that the insurance mortality is still weighted by deaths from disease contracted before the days of better industrial and social hygiene whereas our morbidity statistics have all the benefits of these more modern factors. A further important aspect is that the tuberculosis incidence in any industry will have as its main determinant the expected tuberculosis incidence in the social and economic classes from which it draws its employees. How this may vary in the same race with different economic status is shown by two studies:

Barnard[11] x-rayed a Harlem group of 10,232 Negroes and Porto Ricans, finding an incidence rate of 12,300 tuberculous per 100,000 individuals, to use our previous idiom. Per contra, Ulmar[5] and his co-workers x-rayed 1000 colored applicants for nursing training and found an incidence rate of only 1000 per 100,000. By way of further contrast, in 1935 Bushford and Scott[5] examined 156,000 employees of the English Post Office System, with an incidence rate of 104 tuberculous per 100,000 — a figure less than half that reported here for Philadelphia's industries.

Most industrial physicians in Philadelphia do not treat the tuberculous employee. If the disease is active, immediate contact is sought with either family physician or chest clinic, depending on economic circumstances. This attitude substantiates the concept that industrial medicine is, and should be kept, *intramural*. Our job is the health of the employee *while at work*. The care of tuberculosis is a community responsibility. Our service to that community in this problem lies in the detection of active or arrested tuberculosis through the medium of our examination techniques.

In the industrial prevention of tuberculosis, the plant engineer has outstripped the physician, whose main contribution is the constant recheck of known arrested cases. The engineer has made the modern factory a pleasant, light place for work. The floors are no longer glorified trash heaps. They are thoroughly cleaned nightly and sprinkled and swept four times during the working day. Temperature and humidity are mechanically controlled. The machines are spaced for adequate cubic footage of air per employee, and the employees themselves separated at their various machine stations. Fountains have replaced the common drinking cup.

Where dust is a factor, special exhausts remove it, or the air conditioning velocity is such that dust and fumes are kept at a safe level. Failing this, the employee must wear a mask. In a recent dust count we had the rare pleasure of discovering that every area in our factory save one small space showed lower organic and inorganic particle values than the street air in front of the plant! And we manufacture cigars, which is considered a dusty industry in all the ratings.

THIS report would have been impossible save for the whole-hearted cooperation of many Philadelphia physicians in industry who placed their data at my service. It is my earnest hope that what I am about to say may also have their full cooperation and endorsement.

Philadelphia industrial medicine has an enviable record concerning tuberculosis, but it is admittedly incomplete and can be bettered. The first step is periodic health examinations for our entire personnel. That is well on the way to accomplishment. But the task will never be complete until we can *film* every pair of lungs we examine. This will require either combined paper and standard film, or possibly the new researches of the Moore School of Engineering in standard films will come to our aid. The only present obstacle to the realization of this program is its cost, which most captains of industry as yet see no reason to bear, and the employee certainly will not pay. Public health funds, either municipal, state or Federal, could well be allocated to this purpose. A further practical suggestion would be a mobile x-ray unit

which can be placed at the service of industrial physicians by some central public authority.

But this proposal is merely a part of a wider plan. In the main, a medical department is only present in factories employing 1,000 or more persons; yet 90% of Philadelphia's industries have less than 200 employees. Such establishments cannot afford the services of a physician devoted to their health problems. I suggest that the Committee on Public Health of this College devise a method whereby *all* the industries of this city may be served by physicians conversant with the procedures common to every well organized industrial medical department. Such a step will mean infinitely more industrial physical examinations than are now being made. This suggestion is of singular significance toward forwarding the anti-tuberculosis campaign. It strikes to the very heart of the problem by effecting more examinations, and the more examinations, the more active tuberculosis cases uncovered and the more arrested cases controlled!

In summary:

1. The contribution of industrial medicine to the community tuberculosis campaign is shown to lie in the examination of large employed groups, and the prompt reference to proper channels of those found tuberculous. The necessity for periodic examination in this program is stressed.

2. Suggestion is offered that medical service be extended to all the manufacturing establishments of the community so that these examination techniques may become universal. It is part of this plan that an x-ray chest film shall be considered a routine part of every such examination.

3. The tuberculosis incidence rate in Philadelphia's reporting industries is shown to be better than the incidence rate in the general population of the City of Philadelphia.

4. The beneficent influence of plant sanitation is cited.

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Diagnostic Methods in Acute Upper Abdominal Pain

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THE abdominal emergency brings to every one of us a quickening of interest, and an awakening of personal responsibility to the patient, which is seldom equalled.* Here is a predicament where our patient needs not only accuracy, but also speed of diagnostic methods.

Such methods and their applications are the aim of this discussion which, for the sake of brevity, will be limited to the analysis of acute pain in the upper abdomen.

THE History, usually of such well-recognized value in diagnosis, is frequently slighted in the abdominal emergency because of the inability or indisposition of the patient to talk, and because of the difficulty in conducting a deliberate inquiry with excited friends or relatives. Almost never is the need for action so urgent that careful history-taking can justifiably be neglected, and with tactful persistence the family or even the pain-ridden patient can usually be successfully questioned.

Thus the previous relationship between food-taking and pain in peptic ulcer; the using of lead products in lead colic; the past observation of jaundice or of similar pain in gall-bladder disease; and innumerable other important leads, may be drawn out. Only last month we encountered a patient whose "acute abdomen" was examined by two doctors before diabetic acidosis was detected —the patient himself having been too ill, and too unaware of any possible connection, to volunteer that he was a diabetic.

Much may be learned from the subjective characteristics of the pain. Thus, a very sudden onset suggests a ruptured viscus, or biliary colic, whereas severe pain ushered in by gradually increasing premonitory symptoms is more often associated with small intestinal obstruction, a twisted viscus, or pancreatitis. The abdominal pain of diabetic acidosis usually is preceded by persistent vomiting. Pleurisy or pneumonia gives rise to a more superficial form of pain which is noticeably increased by breathing and coughing. When of cardiac origin, the abdominal pain may merge with the more typical precordial or substernal variety, sometimes with reference to the left arm or neck. In tetany of the parathyroid or hypo-calcemic type, observation of carpopedal spasm provides the indication for a blood calcium determination.

* Read before the Congress of Railway Surgeons at Chicago, September 19, 1938.

The increasing pre-diagnostic use of morphine in abdominal pain has no stronger argument than its aid in enabling the patient to give a coherent history. The fundamentally sound taboo against symptom-masking opiates has probably been abused at times by a blind following of this principle when a properly gauged dose of morphine might have been safely used to increase the value of both history and examination. Needless to say, morphine would not be used in this way except when its need was evident, and then only after a careful preliminary examination followed by continuous observation of the patient, as in the hospital.

PHYSICAL EXAMINATION of the abdomen is hardly a satisfactory or a necessary subject for discussion here. The frequent discrepancies between the location of tenderness and location of the lesion are all too well known to all of us. The absence of tenderness in the presence of pain, of course, fits the picture of biliary or lead colic, or of early intestinal obstruction, or of tabetic crisis, rather than that of ruptured viscus or acute inflammatory lesion.

Sometimes the tumor of intussusception may be felt, and recently we observed a similar tumor which at operation proved to be a mass of strangulated omentum.

Physical examination elsewhere than of the abdomen is another procedure frequently slighted. The most careful chest examination may be required to detect the presence of a lower lobe pneumonia, or of diaphragmatic pleurisy, to account for the pain. Cardiac findings may not, in themselves, be adequate to the diagnosis of coronary disease, but may be present in sufficient degree to suggest the taking of a confirmative electro-cardiagram. It is almost superfluous to mention the need for examining pupillary and tendon reflexes with tabetic crisis in mind.

LABORATORY FINDINGS are often of great help when kept properly subservient to clinical judgment. Leukocytosis, of course, is a common denominator in all of the inflammatory or perforative lesions, with polymorphonuclear cells predominating, and an increase in young neutrophiles. This acute inflammatory blood picture is augmented by an increase in sedimentation rate to about 18 mm. in less than 40 minutes. Leukocytosis is likewise the rule in the acute abdominal pain of diabetic acidosis where the count often exceeds 25,000. Absence of leukocytosis fits the pictures of tabetic crisis, lead colic, abdominal angina, or early intestinal obstruction.

Glycosuria has been observed in pancreatitis as well as in diabetic acidosis, in which latter condition, however, the presence of acetone and diacetic acid furnish conclusive evidence. So completely can diabetic acidosis simulate a "surgical abdomen" that accuracy demands a urinalysis, by catheterization if necessary, before any study is complete.

Among other laboratory findings worthy of mention are the detection of lead in the urine and stool in plumbism, the lowered blood calcium in tetany, and the increased total non-protein nitrogen of the blood in small intestinal obstruction. In the last named, the presence of indican in the urine is suggestive, although we have found it present in peritonitis without obstruction.

X-RAY undoubtedly is the most helpful single adjunct to clinical diagnosis of upper abdominal catastrophes, and most of us feel that we must use it as invariably as we do our stethoscopes. Repeatedly, intelligent use of x-ray can change doubt to assurance, long before the clinical picture is complete.

Intestinal obstruction is an outstanding example of conditions where x-ray can promote speed and accuracy of diagnosis. Roentgenologically demonstrable gas may appear in the small intestine as early as three hours after the onset of symptoms, and this alone offers strong presumptive evidence of obstruction. A few hours later, such an accumulation of gas may assume a central location, with shadows of "herringbone" appearance due to stretching of the valvulae conniventes by distention. Still later, the gaseously distended loops of small intestine take on the more frequently described "step-ladder" appearance, with multiple fluid levels visualized.

Thus, x-ray can contribute to the discovery of an urgent need for surgical intervention long before the classical clinical findings of intermittent pain and persistent vomiting have reached unmistakable development. At times, a pre-operative hint at the location of obstruction may also be derived, when it is remembered that the x-ray demonstration of distended loops of small intestine in the *lower* abdomen usually is due to an obstruction in the terminal ileum; whereas an obstructed jejunum usually produces the picture of distended loops in the *upper* abdomen.

In over 75% of cases of perforated peptic ulcer, x-ray can demonstrate free air in the peritoneal cavity. Since this pneumoperitoneum appears immediately after the rupture, we may again avail ourselves of a means of earlier diagnosis. Usually, fluoroscopy in the upright position is sufficient to reveal an air bubble which has risen to the dome of the right diaphragm, or, if the amount of air is great, to both domes. If no air is seen, the patient can be re-examined, after lying on his left side for an hour or more to favor the escape of air from the usual site of perforation. A further refinement is to take a vertical x-ray film while the patient lies on his left side. This may visualize air in the form of a sickle-shaped area of decreased density between the liver and the right abdominal wall.

The value of x-ray in demonstrating this evidence of perforated ulcer is so generally known that it is hard to understand why there is not more universal use of the method. The patient under suspicion can readily be taken to the fluoroscopy room with no added disturbance while entering the hospital and an x-ray observer of even limited experience can readily detect the escaped air.

In searching for, or analyzing, extra-abdominal

sources of acute upper abdominal pain, x-ray may again be called upon. X-ray films demonstrating the early or central infiltration of pneumonia may be most helpful in supplementing clinical chest findings; and the fluoroscopic demonstration of a lagging movement of the diaphragm or of a cloudy costo-phrenic angle may even precede discovery of pleurisy by physical examination. Even acute coronary occlusion has recently been the object of x-ray study from the diagnostic standpoint, and evidence has been offered that an infarcted area may be detectable by its relative loss of contractility, and lessened convexity of outline, as observed in x-ray.

ELECTROCARDIOGRAPHY is a two-edged weapon in abdominal diagnosis. A few years ago there was need for greater emphasis upon coronary occlusion as a possible source of acute abdominal pain, but today we probably are more apt to over-shoot the mark and postpone needed surgery while we speculate over a doubtful tracing. In our attempt to steer a middle course, intelligent use of electrocardiography is mandatory.

A most important consideration in evaluating the electrocardiogram is the occasional absence of typical evidences of coronary involvement during the first 24 to 48 hours following occlusion. This is thought to be due to the continued ability of the heart muscle to convey electrical impulses, in many instances, until completely deteriorated by prolonged ischemia. The generalization might be proposed that the *absence* of coronary changes cannot be trusted during the first two days, whereas, the *presence* of typical coronary findings is of the utmost significance. If a previous tracing is available for comparison even small variations are to be considered seriously.

From the confusion of varied opinions about these electrocardiographic changes, a few generally accepted characteristics of recent coronary occlusion may be stated:

1. An elevation or depression of the S-T segment from the base line is present in the electrocardiogram. This is often termed a "high take-off" or "low take-off."

2. The S-T segment shows a convex bowing as it joins the T-wave. This is usually described as "cove-shaped."

3. Typically, the T-wave is peaked, with rounded shoulders. Inversion of the T-wave in lead I is highly significant, although this change may not appear at first.

These comments apply to the three standard limb leads. Lead IV, for which one electrode is placed against the precordium, is rapidly gaining acceptance as a source of greater accuracy in detecting, and even localizing, coronary infarcts. However, this phase of the subject is not sufficiently stabilized for a nonspecialized discussion.

Despite the admitted limitations of the electrocardiogram, it can be at times our sole clue to the coronary origin of abdominal pain, before the appearance of cardiac irregularity, friction rub, leukocytosis, or fever. A working knowledge of the method is essential to accurate diagnostic work.

THE following brief case history is appropriate: A man of 47, a railroad shop foreman, was brought to the hospital at 4:00 P.M. by ambulance, following a sudden collapse while at work. He showed an ashen pallor, clammy skin, and evidences of continuous agonizing pain in the epigastrum. The upper abdomen was firm but not intensely rigid. The heart rate was 106 and regular, with no murmurs. The white blood count was 12,000.

This patient had been treated medically for peptic ulcer in 1923, with a recurrence in 1925 for which a gastro-jejunosotomy was performed. In 1933 (three years before the present admission) ulcer symptoms recurred and a diagnosis of gastro-jejunal ulcer was made. The patient received accurate medical management and obtained an apparently complete recovery.

The ulcer background, of course, offered strong presumptive suspicion of a perforation; so a fluoroscopic examination was made during admission to the hospital. No air bubble was seen under the diaphragm at this examination, nor at a subsequent fluoroscopic examination made after the patient had rested in bed on his left side for about two hours.

During this interval no increase in rigidity had appeared and the white count remained the same. Due to the absence of other evidences, coronary occlusion was suspected and an electrocardiogram was made. This showed no strikingly typical coronary appearances.

The patient was made quiet and relatively comfortable by opiates in moderate dosage, and put under careful observation for the night. At 1:00 A.M. he requested a drink of water. When his nurse returned with it he was dead. Autopsy showed a large infarct in the anterior wall of the left ventricle; and healed scars of duodenal and gastro-jejunal ulcers.

SUMMARY: An attempt has been made to highlight some of the details of diagnosis which many of us are tempted to slight in our urge to take action in an abdominal emergency.

The essential elements of a careful history and physical examination are as necessary here as in situations allowing greater deliberation, and these procedures may be enhanced at times by the judicious use of morphine.

Laboratory tests, selected and interpreted by the clinician, have an importance which reaches a peak in the detection of diabetic acidosis; or, more rarely, of hypocalcemic tetany.

X-ray is so frequently a means of hastening and clinching the diagnosis, that free use of this method is most commendable. Unquestionably, the sorry mortality figures for intestinal obstruction and perforated peptic ulcer would show improvement directly proportional to increased frequency of x-ray examinations.

The electrocardiogram can be a reprieve for a

coronary victim otherwise sentenced to death on the operating table. Not one of us who attempts abdominal diagnosis can escape responsibility for a working knowledge of the electrocardiographic changes in acute coronary thrombosis, with the understanding that these changes may sometimes fail to appear immediately after the occlusion.

Discussion:

DR. JAMES E. McLOONE, La Crosse, Wisconsin: Dr. Sippy has covered the subject I think very thoroughly but, as a surgeon, I think there are two points to be emphasized to the benefit of all. He discussed perforating ulcer and mentioned coronary disease. We have in these two conditions a surgical emergency, not a medical emergency, from which differentiation must be made. It is a life-saving measure if surgery is done in the proper case. I think most of us had drummed into our heads that perforating ulcer was a definite emergency and operative intervention should be done as soon as possible. It is my impression that we have more time to operate than we were told, and having more time we are apt to make more correct diagnoses than if we go in blindly.

The x-ray is one of our many helpful diagnostic methods and those in outlying communities, who see cases sometimes after the perforation has occurred, are anxious to get inside that abdomen, where they would be better off if they waited sufficiently long to determine whether that gas was above the diaphragm.

With reference to coronary disease, I think it is well for surgeons to remember that such a thing does occur and that we have occlusion with pain in the abdomen which simulates a perforating viscus at times.

DR. G. E. HEARST, Cedar Falls, Iowa: I had a patient with a ruptured small intestine two weeks ago who apparently made a good recovery; at operation his abdomen was full of pus and bowel content. Two weeks after the operation he started having severe pain in the stomach, really in the epigastrium, for five or 10 minutes, coming on rapidly and stopping rapidly. At the time of the attacks his temperature goes up and his pulse becomes increased 20 to 30 points. The patient is a young, husky country boy without nerves. The pain comes on rapidly and before we can do anything it disappears. That has been going on for four days, extending from five minutes at first to 20 minutes yesterday. Yesterday the boy cried for morphine and was very much distressed. Blood counts have remained the same. We happened to take a blood count 15 minutes before the pain came on and I asked for another an hour later and they were the same; white count around 10,000, having dropped from 18,000 a few days before. It is a terrific pain and after it disappears he goes along all right. It has no connection with eating, and I do not know what it is.

DR. H. IVAN SIPPY, Chicago (closing): The doctor mentions upper abdominal pain some days after operation for small intestine perforation. The pain comes on in the morning. The pain, he is sure, is genuine and very severe. There is no special elevation of the white count. Of course, it would be the height of folly to try to help him make a diagnosis, but I am sure the doctor is thinking of an obstructive process developing along the line of the small intestine which would not be illogical with his history. The onset of such symptoms is occasionally followed by acute obstruction.

IN CONVENTIONS, this year, all roads lead to Cleveland, O. In June, on days from 5 to 8 inclusive, the Hotel Statler in that city will be the scene of the Twenty-Fourth Annual Meeting of THE AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS with THE AMERICAN CONFERENCE ON OCCUPATIONAL DISEASES AND INDUSTRIAL HYGIENE.

Finger Tip Reconstruction

— *A New Operation* —

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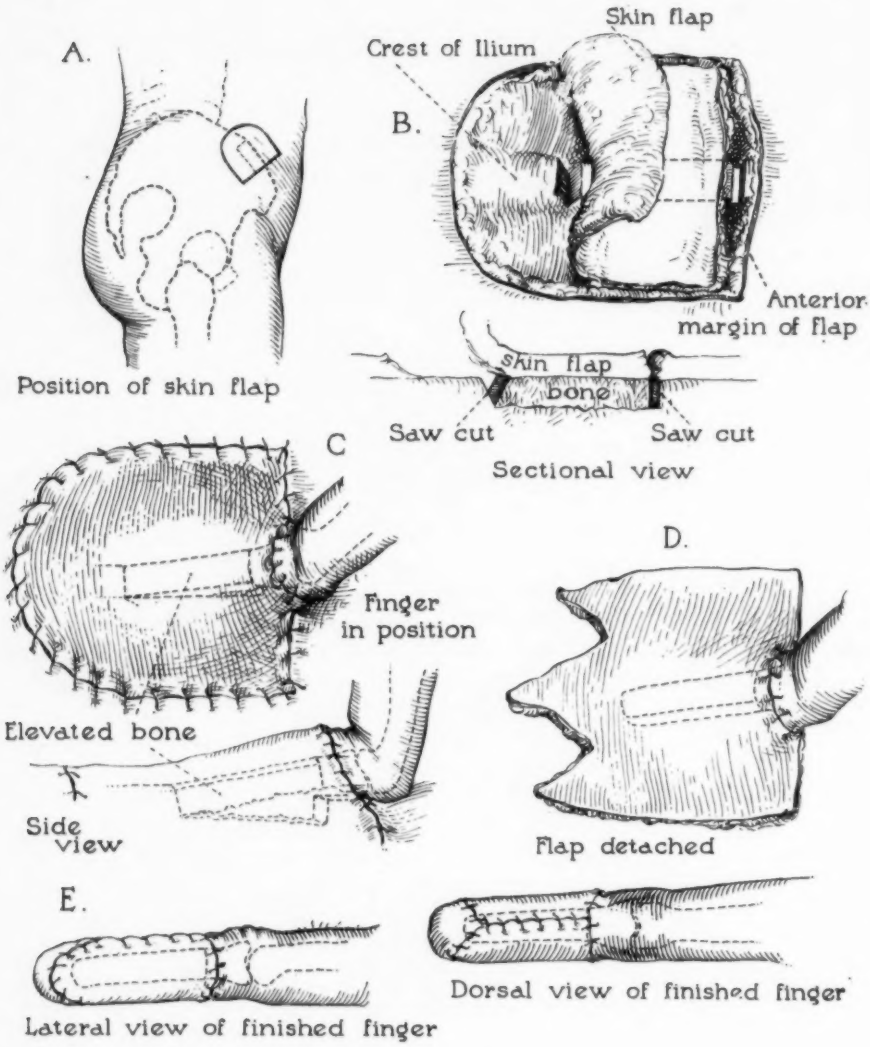
THE loss of the tip of a finger or thumb handicaps an individual in proportion to his need for the normal digit. Not only a physical but a mental handicap may arise, however, from the amputation. In a small percentage of cases there develops during the period of healing a mental state which retards and occasionally prevents the patient's readjustment to his previous occupation. The author has observed the case of a stenographer who, having lost the tip of her principal index finger because of an infection, never fully adjusted herself to this deformity and hence could not type efficiently. The mental upset in such cases is more frequent among women than men. While the great majority readjust themselves in a compartively short time, there remains nevertheless the need of eliminating the handicap when it arises.

Several methods have been advanced for the replacement of a lost finger. The most prominent is that which transfers the second toe to the hand. Although this method furnishes a nail, it presents a tremendous disadvantage in the hand-to-foot position the patient must assume for at least three weeks. The writer was prompted to seek a more satisfactory position for a finger graft.

A working foundation for the reconstruction of a finger tip is found in surgical experience with the transfer of skin flaps and bone tissue. Both are done with considerable success providing a convenient site of supply can be found. In the present case, that of an architect 34 years old, I chose the crest of the ilium on the same side as the digit, which had been amputated just beyond the middle phalangeal joint. A three-stage operation was developed:

1. An area five centimeters posterior to the anterior superior spine was selected and a flap providing ample skin tissue for the restoration was then outlined in methylene blue; the anterior margin forming the base. Along this margin a vertical incision was made to the bone, which was then sawed through to a depth approximately equal to the thickness of the bone needed for the finger. The flap was then undermined and retracted along the posterior border anteriorly without disturbing the periosteum; the remainder of the border was also undercut. Two closely parallel saw cuts were made in the bone at a distance posterior to the first cut corresponding to the length of bone desired for the finger. A small wedge of bone was removed. The rectangular outline for the phalanx was then completed by two parallel chisel cuts leaving bony tissue of apporpriate length and width. A narrow flat chisel was applied at the anterior saw cut to elevate the bone segment sufficiently for apposition against the



phalanx but not so far as to segregate it entirely.

The finger stub was now prepared. A guillotine amputation was made as distal as possible. The end of the bone was freshened and the soft tissue moderately undermined completely around the finger. A dorsal incision was made extending far enough back on the finger to permit the spreading of the soft tissue for suturing to the flap. Hemostasis, of course, was complete.

The finger was flexed and placed in apposition to the elevated bone. The periostea of the abutting bones were sutured and likewise the soft tissue of the finger to the anterior edge of the flap. The undermined flap was sutured in its original site and a pressure bandage of wet gauze and marine sponges applied. The arm was immobilized by a large roll in the axilla, several 10-yard packs and adhesive. This position was maintained throughout the first stage. At the end of eight days the operative site was exposed and sutures removed. The wound was then dressed every second day thereafter. Examination showed bony union at the end of three weeks but the second stage was not attempted until the twenty-fourth day.

2. The second stage consisted of detaching the flap and the grafted bone from the hip, care being used not to disturb their union with the finger. Hot wet packs were immediately applied to the pelvic wound with pressure. The hand was now placed with extended palm down and by measurements previously determined the new bone was shaped and its sharp angles rounded. The flap was now brought around the bone and its edges sutured dorsally along the midline. It was also sutured to the stub. The tip was fitted by removal of wedge-shaped pieces from the end of the flap. Deep, white, fine silk sutures were used, with horse hair superficially. The finger was now mounted on an anterior short splint and the arm manipulated at the shoulder, elbow and wrist. The pelvic wound was undermined liberally, bone wax was applied to the roughened area and the wound closed by usual methods. The patient was advised to support the arm and hand in a sling. Dressings were made every third day.

3. The third stage consisted of correction of the flap's shape and removal of excessive scar tissue.

Local anesthetic was used throughout these operations, but experience would indicate that sufficient gas or ethyl chloride is advisable to permit free manipulation of the arm without discomfort.

CONCLUSIONS. The procedure offers a new technique for the reconstruction of a finger where the middle phalangeal joint is intact.

Favorable points: (1) The site of the operation permits the patient to be ambulatory and comfortable in contrast to the toe graft. (2) The technique provides a smooth and unscarred palmar surface, where the association of the periosteum and soft tissue has been preserved.

Unfavorable points: (1) The operation is not practical where there is excessive fat over the pelvic crest. (2) There is no nail. (3) There are no tendon attachments. (4) Sensation cannot be anticipated in less than six months and during this time caution must be used against abnormal pressure.

Some Unusual Abdominal Conditions

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TO THE general surgeons, the abdominal cavity probably offers the field of greatest speculation and interest.* Even the usual pathological conditions such as appendicitis, and cholecystitis may present such unusual character-

* Presented at the Congress of Railway Surgeons, September, 1938, at Chicago.

istics that the surgeon is unable to be entirely certain of his diagnosis. The strangest and most unexpected conditions may be concealed behind a loop of intestine or seek seclusion under the shadow of the liver, pancreas or spleen.

To further complicate abdominal diagnosis has come an ever increasing frequency of trauma which may cause an acute fulminating condition, or may be responsible for delayed complications.

I have selected a few cases, chosen at random for brief presentation. It is not my desire to bore you with detailed descriptions but rather to present the highlights on these cases which have seemed to me to be somewhat out of the ordinary.

1. C. V., a girl aged 13, tall, slender and very thin; one afternoon was climbing out of a concrete swimming pool in the approved fashion of jack-knifing her abdomen over the edge and pulling herself out with her hands and right foot hooked over the edge. Her foot slipped and she fell back across the concrete edge. She experienced an acute pain in the right lower quadrant of her abdomen. This was quite severe but gradually subsided into a dull ache. When seen some 10 hours later she was suffering with generalized abdominal pain, nausea and definite symptoms of shock, weak and rapid pulse and lower abdominal rigidity. No definite diagnosis was made other than intra-abdominal injury and an immediate exploratory operation was decided upon. Examination showed the right ovary to have been ruptured, in fact it was almost mashed in half, and a profuse and severe abdominal hemorrhage had resulted. The injury was so severe that the ovary had to be removed.

2. S. H., a man, aged 58, reported with a history of gradual loss of weight, no appetite, general inanition, chronic constipation, and a general history of not feeling well. No pain, no abdominal discomfort, no temperature, no increased pulse rate. Examination revealed a definite mass in the right lower quadrant. This mass was sausage shaped, seemed to extend down toward the pelvis. A tentative diagnosis of carcinoma of the cecum was made. X-ray examination showed no intestinal involvement though there was a distortion of the cecum from pressure from without. Urine examination was negative, and there was no history to point to an involvement of the genito-urinary tract, however, the blood chemistry examination showed a marked elevation of the urea nitrogen, N.P.N. and creatinine. Because of these findings this patient was catherized and the catheter left in place for several hours with the disappearance of the tumor mass as the result. We were dealing with a diverticulum of the bladder which was relieved by the V-ing out of a prostatic ring—there being no prostatic enlargement—with the resectoscope.

3. C. B., aged 21. Referred to me after a three week's illness of an obscure nature. Typhoid fever had been suspected. Just prior to entrance into the hospital a mass had been discovered in the left lower quadrant, though not very far below the umbilicus. The mass was not movable and was not circumscribed. A G.I. series was not run because of the evident acuteness of the condition borne out by a high leucocyte count, rapid pulse, evidence of toxemia and continued elevation of temperature together with the additional history of nausea and vomiting, accompanied with abdominal pain. A transposed appendix was thought of, but could not be demonstrated. Operation revealed an abscess cavity pointing below and several inches to the left of the umbilicus. When opened, the cavity was found to extend across and down to the right iliac fossa, a ruptured appendix being responsible for the condition.

4. G. P., a man, 47 years of age, carpenter by trade. Referred after six weeks of illness with a histroy of loss of weight, no appetite whatever; had been jaundiced for three weeks prior to being seen. There was no history of his having run a temperature. No pain or abdominal discomfort. His attending physician had discovered a mass just above the umbilicus, oblong in shape and situated practically across the midline. The sedimentation rate was low. This in conjunction with a stool examination and the other findings, led to the diagnosis of carcinoma of the pancreas. An exploratory operation was performed. The pancreas was found enlarged, almost rock-like in its consistency and non-adherent. Gall-bladder and other surrounding structures were examined. No enlarged gland could be found for biopsy. The abdomen was closed and a hopeless prognosis given. The patient had an uneventful convalescence. This jaundice rapidly cleared up, his appetite returned, the tumor mass disappeared and at the end of three months he was back at work, his health entirely restored.

5. J. S., a boy seven years of age. Sent in to the hospital with a diagnosis of acute appendicitis. He had been seen daily for three days by an excellent diagnostician who reported no rigidity, but some nausea and vomiting, with elevation of temperature during the time mentioned. On the day of admission, there was the fact that two weeks prior to his admission, he had, while playing, run into the edge of a table and had complained for one day of abdominal pain. He had not stopped school. On admission he had generalized spreading peritonitis, with high leucocyte and all the other symptoms. A right rectus incision revealed the peritonitis, but a normal appendix. Further exploration demonstrated an omental abscess almost directly behind the umbilicus, densely adherent to the abdominal wall and to the underlying loop of small intestine which was kinked and partially obstructed. The diseased portion of the omentum was removed and the peritoneal cavity drained. The laboratory reported a staphylococcic hemolytic infection with numerous blood vessels opening into the abscess cavity. This case developed an ileus with obstruction which necessitated a double enterostomy. Two operations were necessary to effect a closure in this case.

6. M. M., aged 34. Suffering with pulmonary tuberculosis. Referred to me with a diagnosis of

right inguinal adentitis. This was of some three weeks duration, growing steadily larger and more painful during that time. Examination revealed the mass, while about the size and consistency of an inflamed and enlarged gland, to lie above and to the inner side of the lymphatic chain. A careful history brought out the fact that the condition had begun with pain, nausea and vomiting and that it had been continuously accompanied by intestinal disturbance. This led to the diagnosis of an appendiceal abscess which upon operation was found to be in the inguinal canal, the appendix evidently having ruptured into the internal inguinal ring.

7. Mrs. A., aged 47. Referred as having probable carcinoma of the uterus. There was a history of her having been "going down hill" for the past four months. Was not seen by a doctor until the day prior to her admission. Examination revealed a large tumor mass occupying almost the whole of the lower abdomen and extending above the umbilicus. This mass was hard, regular in outline and quite tender to palpation. This patient was very deaf and hard to elicit a history from. She had voided since admission and urinalysis showed albumin and casts. It was felt from the consistency of the tumor that the bladder was overlying the mass and she was ordered to be catherized. When I returned in 30 minutes to complete the examination, the nurse was still with her and reported the urine still flowing. I ordered the catheter withdrawn, as 76 ounces of urine had been collected. Three hours later, 36 ounces were obtained by catheter and the tumor mass had entirely disappeared. Added to the bladder paralysis was an intestinal paralysis. An x-ray examination of the spine was ordered, but while being lifted on the carriage, this patient evidently had a coronary occlusion and died in a few minutes.

8. F. H., aged 48. Was referred with a history of repeated attacks of gall-stone colic. A history revealed the fact that he had had his gall-bladder removed 12 years previously in Washington, D. C. A report was had from the surgeon, hospital and pathologist with a history of cholecystectomy for gall-stones and an acutely inflamed gall-bladder. There was no jaundice but the symptoms were so typical of gall-bladder pathology and the patient insisted so strongly that his symptoms were identical with his former trouble that an operation was undertaken. Dense adhesions were found along the gall-bladder fossa, and at the bottom of the fossa was found a small, hard, indurated mass, seemingly the gall-bladder, containing two stones. The common duct was not involved and the cystic duct opened into the remnants of what had been a gall-bladder. Evidently there had been an amputation of the gall-bladder with about one-fourth being left, though it is possible that it may have been a supernumerary gall-bladder, overlooked at the first operation.

These cases are reviewed, not with the idea of distracting anyone's attention from the ordinary and the usual. I have always felt that when in doubt, it is best to choose that condition which is most likely, and not go too far afield in the search for the unusual.

Also, I believe it will be generally agreed that, after it has been definitely determined that an operation is necessary, it is just as well to say as little as possible as to the exact condition one would expect to find.

Clinical Aspects of Cardiovascular Disease

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I THINK you will all agree with me that any adequate discussion of this subject in a period of 15 or 20 minutes is out of the question.* Therefore, all that I can hope to do in this time is to present in an informal way a discussion of some of the more important practical aspects of this subject which are significant in the diagnosis and routine management of patients with vascular decay.

I need pay little attention, I believe, to the importance of the prevalence of vascular disease. It is the greatest barrier to longevity that we have at the present time. More people are destined today to have something go wrong with their arterial system than from any other one tissue. We hear much about carcinoma, but cardiac decay is responsible for more than twice as many deaths as carcinoma. In our own ranks vascular disease takes by all odds the greatest total. In the mortality statistics published by the American Medical Association last year covering the deaths of 30,000 physicians in America, an analysis showed that 70% of the doctors listed had died of something having gone wrong with the vascular system, so that whether you be a specialist, general practitioner, surgeon, or what not, certain it is that 70% of all of you are destined to succumb to some vascular accident. So, wherever we get our statistics, we find not only that vascular disease is killing more people than any other disease but also that the incidence is steadily mounting.

Now this is neither the time nor the place to go into a discussion of what little we know about the cause of human arteriosclerosis. Certain it is that the old adage that a man is as old as his arteries is becoming increasingly apparent. We hear much about wine, women and song, the pace at which we are living, etc., as factors determining how well our arteries hold out. Students of the subject, however, feel that perhaps the most important single factor is the factor of inheritance, so that perhaps the surest way to live long is to select, with great care, your ancestors.

This discussion of the factors determining how well our arteries wear reminds me of a negro

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* Read before the Congress of Railway Surgeons, Tuesday, September 20, 1938, at Chicago.

patient, a white-headed chap who came in for some simple condition and we were impressed by the youthful state of his vascular system. The interns tried to find out his age. They found out that he had been a cook in the Civil War, so he was over 90. Feeling we might learn how to live long, we asked him if he ever used tobacco. He said: "Well, I began to smoke when I was six and I have chewed since I was 12." What about liquor. "I drank all the good liquor I could get." Were you ever married? "Yes, twice." Did he ever have a family? "Well, I had 14 that I knows of." So that was one man's experience, and he had arteries like a young man's.

Now a word concerning the pathology of arteriosclerosis. We know at the present time little more fundamentally about the causes of human arteriosclerosis than did Gothstein, in 1831, who first used this term.

One interesting feature concerning the pathology which is important to keep in mind if we are to appreciate the wide variety of clinical pictures is its very capricious distribution. There is no rhyme or reason, so far as pathology can tell, to the distribution of arteriosclerosis. As clinicians interested in growing and living people, we try, of course, to associate the pathologico-physiology which determines the symptoms the patient complains of with the anatomical process, and to do this we must appreciate this capricious distribution. Why do one man's arteries go to pieces in the cerebellar distribution? Another man's arteries will hold out in the brain, but the coronary arteries have failed. Another man's coronaries and brain arteries may hold out, but the renal arteries may be the seat of widspread disease.

Now ordinarily the functional significance of vascular decay is that it will determine the symptoms that the patient has and will determine the clinical picture that they present on examination, and I would call your attention here to three vital structures, three vital organs. A failure of the circulation of these structures determines to a large extent the symptomatology and clinical picture. These organs are the brain, heart, and kidneys. The circulation from the physiological standpoint, or considered from the view point of a physiologist, is designed—in fact the whole apparatus is designed—to maintain a steady flow of blood through microscopic channels. We have a heart or central pumping station, the veins, the arteries, the arterioles and the capillaries where the real business of life takes place. As we age, we all know that some time after the age of 35 or 40 we will show evidence of arteriosclerosis. On the other hand, whether that arteriosclerosis gives you any symptoms will depend, to a large extent, upon the rate at which the process advances and where it is located. For example, we may do a postmortem on a man of 85 who died of pneumonia and find extensive, serious involvement of the large arteries, such as the aorta, the branches from the arch, and yet this individual had no symptoms from that process. On the other hand, we may do a postmortem on a man who thought he was in perfect condition and find in the coronary system a serious arteriosclerosis and a fresh thrombus.

The region of the vascular system may be responsible, so that functional significance of the process depends upon where it is located. If it gets a stranglehold on the blood supply, which is a vital organ, we may have serious symptoms and death.

NOW something about hypertension. Recent work, particularly that of Goldblatt, has revitalized, as it were, the whole conception of Richard Bright who postulated that the changes in the arteries, the thickening of the left ventricle in patients dying with scarred kidneys, was due to retention in the blood of some substances which the kidney did not eliminate and caused resistance of the flow of blood to the periphery. Remember that Bright knew nothing about high blood pressure in 1825, so for 100 years we have been puzzled about the problem of hypertension. Very recently, due to Goldblatt's work, evidence is increasing to show that vascular disease, or that an interference experimentally to the blood supply of the kidney, causes persistent elevation in the diastolic and systolic pressure in dogs and monkeys. This is the first time it has been reproduced in any animals, and it is not too much speculation to say that an individual dealing with vascular decay may or may not have hypertension. If the disease affects the renal vascular system, and primarily the smaller renal arteries, that patient has hypertension. If the process happens to spare his kidneys, he may go on and develop extensive arteriosclerosis elsewhere in the body without ever having hypertension. Thus we see patients getting on in years who have sclerosis of all the accessible arteries but who never run a hypertension. You have all seen such men and women sometimes so sclerotic and calcified that you feel sure they cannot live long, and yet those people survive without hypertension. We see people on in years who have what we might call systolic hypertension, with a systolic of 180 to 200, but whose diastolic pressures are not elevated. A man may have a systolic pressure of 180 and a diastolic of 95 to 100. That man may survive to an older age than a patient with a correspondingly elevated diastolic, indicating, we believe, that this latter patient with diastolic elevation has renal arteriosclerosis.

Now to return to the clinical picture and the more practical aspects of management. We have seen that if the process spares the kidney the patient may have no hypertension. That does not mean that he may not be destined to develop symptoms and die of a vascular accident. We see patients with an angina picture, etc., with and without hypertension. We see them with cerebral accidents, hemorrhage, etc., without hypertension. But you do not see people dying in uremia from vascular disease without hypertension. Those patients have hypertension.

Statistically what do we find in postmortem examinations of a large series of unselected cases?

We find the majority with hypertension dying of cardiac failure with or without a coronary accident, with or without the angina picture. Second, we find cerebral accidents, thrombosis or hemorrhage, and last in number, around 8 to 10%, people dying in uremia. So, assuming that a patient beyond 50 who may be dealing with vascular decay comes to you, the present-day informed clinician looks upon this patient as an individual whose vessels are not holding out and who is destined to develop, statistically speaking, one of the three accidents mentioned. Occasionally you will see one who is able to survive two major accidents and die of cerebral hemorrhage. I have seen just one man who survived long enough to have a blow-out in all three vital areas. That situation is very rare.

IN THE routine physical examination of an individual of 50 or more, in whom we feel we are dealing with a vascular disease, what do we look for? In the first place, we try to evaluate as well as we can how well this man's vessels are holding out as refers to the three vital areas. One can, for example, in the ordinary history discover whether his capacity to think straight is gradually waning. We all know that the man beyond 50 with cerebral arteriosclerosis may have character changes and in five to 10 years may have the intellect of a 10-year old child and may live on to the age of 75 or 80.

Then there is the clinical picture of the cerebral hemorrhage of Wallace.

Now about the heart. The cause of mounting indidence of heart failure is due to hypertension and to the mounting incidence of arteriosclerosis. Studying patients with hypertension at postmortem reveals that 60% have significant coronary arteriosclerosis, so one keeps that fact in mind in dealing with patients with hypertension. At the present time we have no accurate way of determining the exact state of the coronary arteries. We have to do this by going into the history of the patient to find out if, when he loads his heart, he has any evidence of an angina picture. Secondary changes in the myocardium of a fibrotic nature may be revealed by a pathologic electrocardiogram. But the informed clinician anticipates that the patient with hypertension about 65 times out of 100, will develop signs of a balky heart. That may happen with or without coronary accident. So suppose you are dealing with locomotive injury with hypertension. In the ordinary examination you will do the best you can to find out about his capacity to think. You will examine him carefully for evidence of myocardial insufficiency; breathlessness on exertion; anginal attacks, or such early signs of left ventricle failure as cardiac asthma. The man may think he is all right, but has some asthma. You go into that history and find that when he sleeps at night he is awakened with a sense of suffocation; he cannot get enough air, and then goes back to sleep. That is an early symptom of block in the left ventricle which may be the earliest manifestation for several weeks. That may happen long before the stage of edema.

Now, examination of the kidney function in a patient with hypertension is important. We have heard a great deal lately about kidney function tests. They have become increasingly complicated so that at the present time you put your patient through the mill with considerable expense, with elaborate chemical tests that throw no more light, in my opinion, than some very simple test. I refer to the capacity of the kidney to concentrate urine. A very simple test is to have the patient eat the evening meal and no more food or water until next morning. He voids at bedtime and brings in his morning specimen. If he does not have heart failure, we want to know what his function is, and if he can bring you urine with a specific gravity of 1025, you can forget about renal damage. If it is 1020, he has lost some power to concentrate, and if it is 1010 he is in a bad way and will develop uremia at any time. That is a simple test that throws all the light that the practicing physician needs to throw on the problem of renal function.

After all these tests have been done and we have as accurate a survey as we can get of the state of his arteries as they relate to these vital organs, then the next problem is what we will do about it. We know we have no medicine at this time that will turn back the hands of time; in other words, we have no specific for the aging process, and since arteriosclerosis appears to be closely associated with the aging process, it is obvious that we are not going to cure our patient. On the other hand, there is much we can do to see that he spares himself as much as possible and in that way, perhaps, his machine will stay on the road. I think it is very important to put a big responsibility for their future in their own lap. It is my habit to say to a man who is 55 years of age: "You know as well as I do that I am not going to give you a new machine or new parts. Your concern is to keep this machine on the road as long as you can. It is up to you to learn to run this machine a little more carefully, and do not think you can perform with it as you did many years ago. That is out of the question. A large part of your future will depend upon how well and carefully you run it from now on, and I do not want you to leave the office thinking I will give you new parts to make you younger." Then I give them reasonable advice as to diet. Many of these patients are overweight and reasonable restriction may help them reduce, and with that may come some drop in pressure. Any factor that loads the heart, as effort, emotion, or overeating, should be, of course, guarded against, and you can explain to them that restlessness is the most reliable single symptom that they are loading the heart more than the blood supply warrants. There is no group of patients who are more victimized than those with vascular disease.

Discussion:

DR. FRANCIS D. MURPHY, Milwaukee: The problem of hypertension to any physician is an important one, but I feel that to railroad physicians and surgeons it is doubly important. For not only is there the responsibility to the patient, but there is also the responsibility to the doctor of the railroad in making just decisions.

Dr. Scott presented the more practical points in a very able way. From his work in the clinical and pathological phases, he is capable of giving this, so I shall just emphasize a few of what I think are significant things in the practical work with hypertensive individuals. One of the important things that must be kept in mind is that the height of blood pressure is not always the most significant thing. If the diastolic pressure is high—120 or above—it tends to be fixed and, of course, that is very important. After all, hypertension is an identification mark that something is wrong with the vascular system of the body, but there are, as Dr. Scott has said, three most important organs influenced by the changes. When the patient with hypertension comes to your office, you should make sure that the hypertension is one of the fixed type and not one that is dependent upon some nervous influence and will drop after a few days or weeks. When this patient comes in, I want to emphasize that he may not have hypertension. I remind you of the importance of taking the blood pressure on both arms for sometimes there may be marked hypertension on one arm and none on the other.

When we come to study this, the heart is the center of our problem and the size of the heart is an important thing. We have, I believe, paid a little too much attention to the laboratory tests. Although I know these are important, yet I find too many doctors capable of estimating the size of the heart, the rate and the rhythm and those other simple things are passing them over and spending too much time on electro-cardiograph tracings. After all, a patient with hypertension is a candidate for heart failure, and when we look at the practical side, this individual will have some pain in the chest or will have some shortness of wind or some swelling of the legs or some edema. Those things should be looked into and the size of the heart taken into consideration, and you can tell by stethoscope the duration of the diastole and systole and draw some definite conclusion.

I shall not say more about these very important things except to emphasize the importance of an eye-ground examination. I believe that everyone doing this work with hypertension should learn how to use the simple ophthalmoscope, because the eye grounds often give us an idea of what the kidneys are doing and what the blood vessels of the brain are doing. Finally, when the patient with hypertension comes in, the kidney is very important to investigate, and do not forget that albumin in the urine, a few pus and red cells may mean more than just a little vascular disturbance. There may be stones in the kidney that have an influence on the hypertension, and sometimes an x-ray examination or careful study will reveal renal stones and, furthermore, the crushing of these stones often will be followed by a very marked drop in the hypertension and the patient will appear to be quite well.

QUESTION: I should like to ask concerning surgery in vascular disease?

DR. ROY W. SCOTT: This is a big order. It is impossible to say very much about any of it. Regarding the surgical management of hypertension, about which we have heard a great deal, it seems to us who have spent our lifetimes practically in trying to learn something about vascular disease that the surgeon, having exhausted his legitimate field of endeavor, is getting over into the field of medicine in trying to combat the mounting incidence of hypertension. All I can say is that any surgical procedure which one way or another increases the vascularity of the kidney may do something in the way of lowering the patient's blood pressure, but as to this haphazard cutting of nerves that are not designed to increase the vascularity of the kidney I can see no justification for it, so I do not anticipate any great revolution in our treatment of hypertension by surgical means. Furthermore, we deal with the problem of human arteriosclerosis, and in examining the kidneys of many people dead of hypertension one becomes increasingly impressed with the extent and severity of the arterial disease in the renal disturbance.

QUESTION: I am interested in the blood pressure in vascular disease. I have a patient with a systolic which is not very high but the diastolic pressure is down to 30. How is this explained.

DR. SCOTT: The problem of low diastolic pressure in arteriosclerosis is interesting. I have seen several instances of patients with a systolic of 180 and a diastolic of 70, 60, 50 or 40. In such cases I have been able to prove that they have serious arteriosclerosis of the aorta. The aorta has two functions: One to act to convey blood to the periphery; second, it has a very interesting function as a dampener or shock absorber taking up the shock of the ventricle systole. The aorta dilates and prevents that shock from going out to the periphery to the vessels less able to stand it. Histologically it is designed for this purpose. In individuals with extensive arteriosclerosis of the aorta, in so far as it is sclerotic, it is less elastic and is like a rigid tube and that shock will be sent to the periphery. In such patients with low diastolic pressure I would want to investigate the aorta very carefully, and fluoroscope or x-ray may show calcified aorta.

Now, I entirely agree and want to thank Dr. Murphy for pointing out the significance of eye-ground examinations in all patients with vascular disease. I often say that if I were deprived of all instruments of precision, I would hold on to the ophthalmoscope as the most valuable single instrument of precision in estimating the condition and prognosis in a patient with vascular disease, and I agree that all practitioners should learn to use it, since the retinal arteries are the only arteries in the body we can see.

The question of Buerger's disease in vascular disease is a big one. The cause is not known. I would not attempt to try to cover the question of peripheral vascular disease. Its detailed management, of course, requires much more time than we have available this morning.

Treatment of Low Back Pain

—Observations as to the More Common Origin of "Sacro-iliac Disease"—

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THE common affliction of low back pain and sciatica is of great social and economic importance.* The illness is often instantly and totally disabling, and frequently develops into a chronic, relapsing and very painful disease. Prompt relief from pain must be given to avoid the early appearance of permanent postural changes due to spastic shortening of the muscles and to inflammatory changes of the fasciae and ligaments.

The infrequent evidence of pathology in painful backs, and the frequent presence of bony variations in symptom-free backs, suggest a re-examination of the long accepted theory on the origin of low back pain and sciatica. The old conception is based on the assumption that mechanical pressure caused by structural changes is the prevalent factor responsible for the pain. Logically the next step seemed to be an examination to determine the existence of a neurological etiology for many cases of low back pain and sciatica which reveal no gross x-ray pathology. A primary neurological origin, or the presence of minor pathological changes, might be accepted if the pain could be eradicated by direct treatment to the affected nerves.

A review and discussion of the topographical

* Read before the Medical Conference of General Motors Physicians, Flint, Michigan, May 20, 1938.

anatomy and physiology of the nerves of the lower back, as well as an evaluation of the x-ray findings in painful backs should help to clarify the problem and point to a more effective and simple treatment.

Many phases of the pain mechanism are positively known, while others have theoretical foundation only. To understand a specific pain problem one must know the origin of the pain and its radiation. The mixed spinal nerves of the back are formed by the union of the anterior motor spinal root fibers with the posterior sensory nerve fibers out of the spinal ganglia. (Fig. 1).

The spinal ganglia of the lumbar nerves are located in the intervertebral foramina. The ganglia of the sacral nerves lie in the sacral canal.

From the spinal nerves arise the weaker posterior and the stronger anterior branches. The posterior branches of the lumbosacral nerves supply the deep muscles of the lower back, the intervertebral ligaments, the ligaments of the pelvis, aponeurosis and periosteal attachments, the lumbo-dorsal

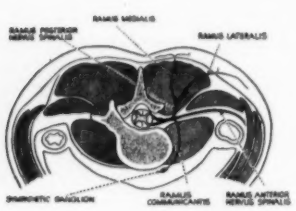

Fig. 1.

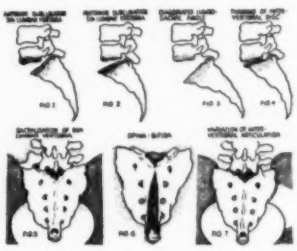

Fig. 2.

Fig. 1. Cross section of lumbar region showing spinal column with anterior and posterior rami of spinal nerve. Fig. 2. Anatomical variations of the "X-Ray Variable" group

fascia, a portion of the gluteal fascia, the intervertebral and sacro-iliac articulations and the skin of the lower back and gluteal region.

The anterior branches of the lumbo-sacral nerves form the strong plexus lumbo-sacralis. They supply most of the muscular structure of the abdominal wall, the pelvic muscles and the muscles and skin of the lower extremities.

For the purpose of diagnosis and the choice of therapy a new x-ray classification proved of great value. We were able to select a large group of back cases which reacted favorably to a new, conservative treatment where previously these cases had presented considerable difficulty in handling. Some of these acute and chronic painful backs were of traumatic origin, while others had no traumatic history. In our classification all back cases were divided in an "x-ray positive," an "x-ray variable" and an "x-ray negative" group, with borderline cases placed in the more conservative group.

The X-Ray Positive Group

1. Fractures of the bony structure of the back including Kümmels disease.
2. Inflammatory and neoplastic pathology of the bony structures of the back.
3. Changes affecting the intervertebral disc. (Herniation of the nucleus pulposus, extreme thinning of intervertebral disc.)
4. Tumors of the spinal cord.
5. Dislocations of the vertebrae and sacro-iliac joints.

The x-ray diagnosis is usually simple and conclusive, but may require a special x-ray technique if herniation of an intervertebral disc or pressure from a spinal cord tumor is to be confirmed.

The X-Ray Variable Group

1. Anterior subluxation of the 5th lumbar vertebra.
2. Posterior subluxation of the 5th lumbar vertebra.
3. Exaggerated lumbosacral angle (average normal 42.5 degrees).
4. Moderate thinning of the intervertebral disc.
5. Sacralization of the 5th lumbar veterbra (unilateral or bilateral).
6. Spina bifida (hemi-vertebrae, congenital).
7. Anatomical variations of the posterior articulations of the lumbar vertebrae. (Fig. 2.)

These structural changes most frequently present anatomical variations, rather than a pathological state. Therefore, this group was named the "x-ray variable" group.

More x-ray variations are found in the lumbosacral and sacro-iliac region than in any other part of the back. Statistics, however, show that at least

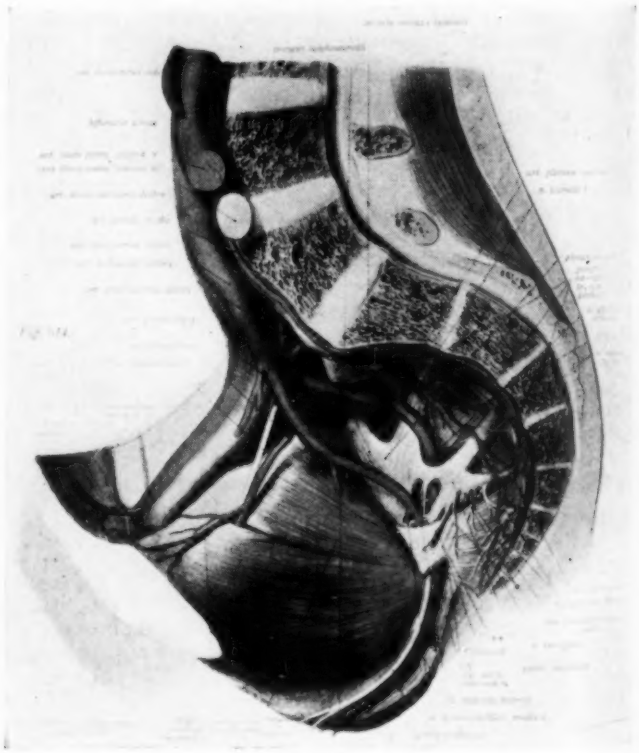

Fig. 3.

Sagittal section showing weakest part of the spinal column. (From Joh. Sobotta, Lehmann's med. Atlanten, Bd. IV 548)

50% of the spines with structural abnormalities in the lumbosacral region are symptomless.

The lumbosacral region is structurally the weakest part of the spinal column. (Fig. 3.) Slipping of the 5th lumbar vertebra, destruction, or marked proliferative changes may logically cause narrow-

ing of the lumbosacral intervertebral foramin and in extreme instances produce pressure on the 5th lumbar nerve which is the largest of the spinal nerves and passes through the smallest of the lumbar intervertebral foramina.

The posterior branches of the 5th lumbar nerve supply the ligaments, fasciae, muscles and the posterior intervertebral articulations of the inferior part of the back. Irritation of the posterior branches of this nerve may cause low back pain.

The anterior branches of the 5th lumbar nerves, together with parts of the sacral plexus, make up the sciatic nerve, the largest nerve trunk in the body, and inflammation or pressure on the anterior branches may produce sciatica. If the irritation affects the spinal roots, or both the anterior and posterior branches of the lumbar and sacral nerves, low back pain and sciatica may result.

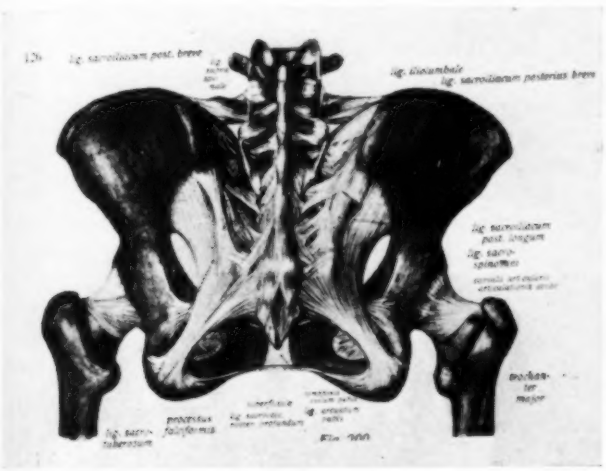

Fig. 4.

Posterior view showing lumbosacral attachments. (From Joh. Sobotta Lehmann's med. Atlanten, Bd. II, 126)

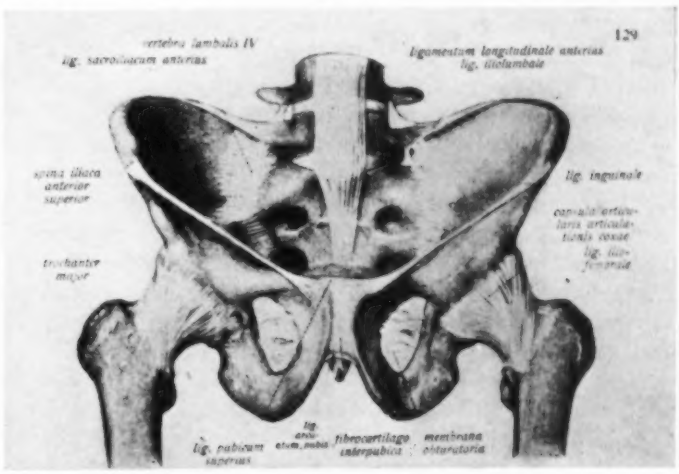

Fig. 5.

Anterior view showing powerful sacro-iliac attachments. (From Joh. Sobotta, Lehmann's med. Atlanten. II, 129)

It is a widely accepted fact that the lumbosacral posterior articulations may be damaged through arthritis, subluxation or even acute or chronic stresses and thus cause low back pain.

Careful diagnostic judgment must be used in interpreting roentgenograms in the "x-ray variable" back group.

The X-Ray Negative Group

The "x-ray negative" group includes all cases of painful backs and sciatica without abnormal x-ray findings. The etiology may be traumatic, infectious, neoplastic, even functional, or due to pressure from adjacent organs. The reason for the pain may be found locally, it may be referred, or transmitted from any part of the body.

The main representatives of this group are:

1. Strains, sprains and bruises of the soft tissues and joints of the back.

2. Inflammatory changes of the soft tissues, nerves and joints of the back as in myo-fascitis, fibrositis, neuritis (sciatica), arthritis. (The inflammation may be local or the result of a distant focus of infection, unspecific or specific as: tuberculosis, syphilis, osteomyelitis, typhoid fever, etc.)

3. Referred back pain from organic disease, as: Kidney, bladder, prostate, vesiculae seminales, pelvic organs of the female, intestinal tract (rectum-coccygodynia).

4. Statural (flat feet) and postural (extreme spinal curvatures, stooped shoulder) conditions.

5. Functional spines in asthenic and neurotic individuals.

Sacro-iliac Syndrome

ANY one or a combination of these "x-ray negative" back conditions is often conveniently named "sacro-iliac strain," "sacro-iliac sprain," or just "sacro-iliac disease." Minor bony changes in the sacro-iliac region are the rule rather than the exception, and only comparative x-rays before and after the onset of sacro-iliac pain should be considered of indipustable diagnostic importance. It is felt that roentgenograms furnish a valuable diagnostic check but should never be considered as sole diagnostic proof, where minor changes in the sacro-iliac region are found.

Sacro-iliac strain or sprain is the most popular term used for back pains with or without sciatica. This diagnosis had been accepted since Goldthwait and Osgood in 1905 described a symptom complex supposedly caused by relaxation, subluxation or arthritis of the sacro-iliac joints. The back pains were believed to be referred from damaged ligaments, affected sacro-iliac joints or even irritated lumbar nerve trunks (of the 4th and 5th lumbar nerves). In medical practice this misnomer has been as popular and as vague as the term "nervous breakdown." Medicolegally it has proved a universal costly excuse for all kinds of low back pain.

No joint in the body is as firmly built as the sacro-iliac joint. Its bones are stout, and being almost in an even plane, there are no exaggerated angles to be easily disturbed. The supporting ligaments, anteriorly and posteriorly, are firm and rather wide and short, and the protecting long back muscles are among the strongest in the body. Only a few degrees of sliding or rotary motion are pos-

sible and slips and strains can hardly occur. It is difficult to believe that the slight subluxation, if it should happen, could cause severe referred pain.

Architecturally, the sacro-iliac region is the most ideally constructed part of the back, while nature has treated the lumbosacral region poorly. (Figs. 4 and 5.) The rather unstable lumbosacral joint with its frequent anatomical variations is much more susceptible to strain, than any other part of the spinal structure. Logically, this joint would be the first to suffer from any trauma to the lower back region. In the rare instances where gross subluxation of the sacro-iliac joints occurs, in cases where extreme degrees of arthritic changes existed, or where tuberculosis and osteomyelitis produced severe pathological changes, the reports speak of little pain. Roentgenograms which show some density in the sacro-iliac joint spaces, or even arthritic lipping must be cautiously interpreted as to the causative factor of low back pain and sciatica. It is difficult to conceive how sacro-iliac pain is produced through irritation of the lumbosacral nerve trunks, since these lie loosely and anteriorly upon the joints. Even the fact that sacro-iliac joint fusion in some instances has given symptomatic relief does not prove the old sacro-iliac pain theory. One should assume rather that the severe surgical procedure would more often irritate than benefit. Probably the relief obtained is frequently due to destruction of pain producing areas in the course of the operation.

Treatment

THE "X-RAY POSITIVE" GROUP:

The "x-ray positive" group always requires competent orthopedic treatment. No attempt shall be made here to discuss proper surgical, neuro-surgical or specialized orthopedic procedures.

THE "X-RAY VARIABLE" GROUP:

The trend in the treatment of painful backs belonging to the "x-ray variable" group is more and more conservative. The radical measures formerly used were based on the conception of lumbosacral and sacro-iliac disease which is now questioned by many careful observers. Conservative treatment always should be attempted and will in most instances lead to a permanent cure. Only severe and unresponsive cases require more radical orthopedic procedures.

THE "X-RAY NEGATIVE" GROUP:

Conservative methods always should be employed in the treatment of painful backs of the "x-ray negative" group. We would digress too far in describing those helpful procedures, such as temporary back supports, improved physiotherapeutic procedures or to list the external and internal medications used in the treatment of low back pain. It is our purpose in this study to present at length a simple, effective treatment for painful backs which will give symptomatic relief from pain in many "x-ray positive" backs, will make permanently symptom free many backs of the "x-ray variable" group, and will cure still a greater number of the "x-ray negative" group. It is logical that a symptomatic treatment which will give quick relief can lead to lasting recovery only if the predominating groups of painful backs are caused by passing difficulties such as stresses, strains, subluxations, minor tears and inflammatory conditions. Pathological changes caused by fractures, dislocations, severe structural abnormalities, or destructive inflammatory and neoplastic disease can not be permanently relieved by this method.

It was felt that low back pain and sciatica in the "x-ray variable" and in the "x-ray negative" group were seldom caused by structural embarrassment but more often by inflammatory irritation of the back muscles and fasciae, the lumbar nerve roots, lumbar nerves, or their branches. Even when severe pressure was present it was suspected that the pain frequently was due to inflammatory swelling of the protective tissues surrounding the nerves, rather than the result of pressure from encroaching bone structures.

The abundant ramifications of inter-communicating nerve branches render it almost an impossibility to ascertain the exact location of the focus of a certain back pain or sciatica. An irritation in the lower back region (posterior nerve branches) may cause referred sciatica, while irritation of the sciatic nerve (anterior nerve branches) may produce referred low back pain. We have been able to observe a case of sciatica originating as high up as the 9th and 10th thoracic nerve roots. (Fig. 6.)

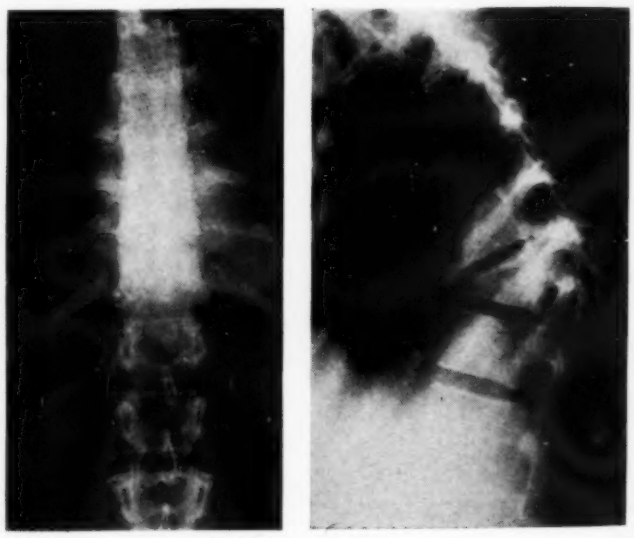

Fig. 6.
Case of sciatic pain caused by compression of 9th and 10th thoracic segments

Believing in a theory which favors local and referred irritation of the nerves of the back as the prevalent etiological factors producing low back pain and sciatica, we attempted to devise a simple, harmless method which would give immediate relief from pain for a prolonged period and, if possible, lead to a permanent cure. It was reasoned that a therapeutic approach, affecting the muscles and fasciae, the larger nerves and their ramifications directly, would be successful. The symptomatic relief must be of such duration as to allow nature and the physician to remove systemic in-

fection, local inflammation, or the result of traumatism, responsible for the pain. Muscle and ligament spasm must be relaxed by the procedure, avoiding pulling and pressure on the affected parts and thereby hastening the healing process. It is industro-medically and psychologically of paramount importance to prevent early postural changes.

As far back as 1934 I attempted to attack the problem of low back pain by injection of 5 cc. or more of 0.5% to 1% Procain solution deep into the low back muscles. It was hoped that the posterior branches of the lumbosacral nerves would be reached and that the anesthetic effect would perhaps be carried to the ganglia and the posterior nerve roots. The immediate results were most striking, but the pain-free period was too short. The injections were first made at the point of greatest tenderness, but the selection of the area of injection was soon narrowed down to a region, low in the back, later to be described.

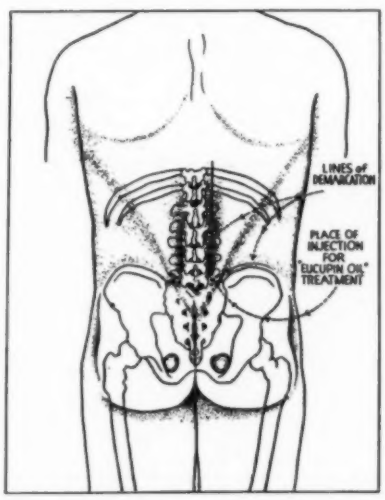

Fig. 7.
Diagrammatic sketch showing location of site of injection of Eucupin Solution in Oil

In the search for a preparation which would provivde both immediate and prolonged relief of low back pain, the local anesthetics in oily solution were considered. Eucupin Solution in Oil was suggested to us by Dr. M. M. Marks. This solution had the following formula:

Eucupin Base	.1
Ethylaminobenzoate	3.0
Benzyl Alcohol	5.0
Oil Sweet Almond q.s.	100.0

Eucupin (isoamylhydrocupreine), a derivative of the alkaloid, cupreine, possesses superior anesthetic qualities, and is strongly bactericidal. The lasting action of the anesthetic may be explained not only by the slow absorption of the oily solution but also by the fact that Eucupin remains locally in the tissues for quite some time and has been recovered even four days after injection.

We have been using Eucupin Solution in Oil exclusively since November, 1936, for the perineural treatment of low back pain.

Technique

WITH the patient lying on the abdomen the sacro-iliac region is surgically sterilized with tincture of merthiolate. A 5-cc. syringe with a 1½ in. 21-gauge needle is used for the treatment. To locate the point of injection the course of the lumbar spine and the crest of the ilium posteriorly are palpated. A line is drawn parallel and 1 in. to 1½ in. lateral (according to thickness of spine) to the spinous processes of the vertebrae. A second line is drawn along the upper border of the ilium and continued posteriorly until the two lines intersect in the lumbosacral region of the back. This is the point of injection. (Fig. 7.) The needle is inserted vertically to its hilt with a quick pressure, the plunger of the syringe is withdrawn slightly, and if no blood is aspirated, the injection of 2 cc. of Eucupin Solution in Oil is begun.

The greater amount of the oil is deposited in the deeper parts. The injection is continued as the needle is withdrawn and should be completed when less than one third the length of the needle is above the surface of the skin. This procedure obviates the injection of the oil solution into the superficial tissues.

The parts should be massaged vigorously after the injection. The needle does not endanger vital structures on full insertion. (Figs. 8 and 9.)

The procedure causes very little discomfort and the immediate result seems almost miraculous. The patient who experienced excruciating pains on motion and who had to be helped on the table, will step down unaided and may be entirely free of pain. In most instances no more discomfort is felt on motion or on bending, although a slight feeling of numbness may be present at the site of injection. A few cases had to be reinjected a day or two later and one case one week later to effect a permanent cure.

In those cases in which the pain-free period was short it was believed that a diffuse myo-fascitis was present and that the injection into the small area did not reach the major superficial parts of the affected back muscles. Where the pain recurred as late as one week, we suspected irritation of the posterior intervertebral articulations and their capsule, and the pain-free period probably was not long enough to accomplish healing of the painful focus. In these cases a short rest in bed is indicated, and in most instances application of heat to the back and mild pain relieving medications were suggested, but seldom used by the patient.

Fig. 8.

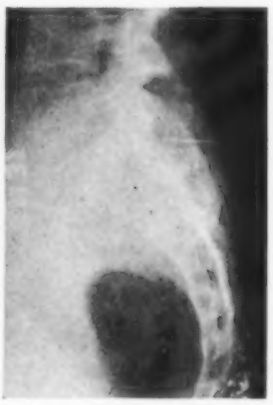

Fig. 9.

Fig. 8. Posterior anterior roentgenogram showing injection needle in position. Fig. 9. Lateral roentgenogram showing injection needle in position

In more than 200 cases treated with Eucupin Solution in Oil since November, 1936, no undesirable after effects were observed. Since that time we have used the Eucupin injection treatment routinely in all cases of acute or chronic low back pain. If sciatic pain accompanied low back pain oil injection was tried first and in a few cases of low back pain with mild sciatica, the sciatic pain also could be relieved by this procedure. However, in most cases of sciatica, perineural injection of the sciatic nerve with 100 cc. of 0.1% B-Eucaine in normal salt solution was necessary to free the patient from pain.

Thirty-five consecutive, unselected painful back cases from one industrial plant in Kansas City, Missouri, were treated with Eucupin Solution in Oil injections after November, 1936, during a period of about six months. These cases were compared with records of 35 consecutive back cases from the same plant, treated before that time, and, without Eucupin Solution in Oil injections. It was found that the disability period in the oil treated cases was reduced by 59%. In three of the 35 Eucupin Solution in Oil injected cases some pain recurred in from two to seven days. Reinjection eliminated all pain permanently and there was no further loss of time in the three reinjected cases.

In order to learn the distribution of the oil in the tissues, after injection, it was necessary to use a radiopaque oil. The manufacturers of Eucupin were asked to produce a Eucupin Solution in Oil containing 20% iodine. This was accomplished and we were furnished with a Eucupin Solution in iodized oil with the following composition:

Eucupin Base	.1
Ethylaminobenzoate	3.0
Benzyl Alcohol	5.0
Iodized Oil (20% iodine) q.s.	100.0

After the iodized oil was found to be non-irritating to the tissues, it was used for back injections, employing the above described technique. The iodized oil was painless on injection and produced a well defined x-ray shadow. (Figs. 10 and 11.)

Anterior-posterior roentgenograms one week after injection show the iodized oil deposited in a streaky fashion as far down as the third sacral vertebra and in some cases as high as the third lumbar vertebra. The width of the oil shadow was from 1½ to 2 cm. in an oblique plane while the lateral x-ray view showed a sagittal distribution of the oil from 2 to 3 cm. in width.

Another series of roentgenograms taken one week after unilateral injection show the anesthetic oil as low as the coccyx and as high as the third lumbar vertebra. Due to the increased distribution and also to probable absorption the x-ray shadow becomes thinner and less distinct. (Fig. 12.) The absorption is apparently very slow since a distinct x-ray shadow of the iodized oil may be observed after more than six weeks.

Fig. 10.

Fig. 11.

Fig. 12.

Fig. 10. Anterior-posterior roentgenogram after bi-lateral injection of Eucupin Solution in iodized oil. (20% I.) Fig. 11. Lateral roentgenogram after bi-lateral injection of Eucupin Solution in iodized oil. (20%.) Fig. 12. Lateral roentgenogram one week after unilateral injection of Eucupin Solution in iodized oil

Summary

OUR observation and successful treatment of many hundreds of painful back cases during a period of more than 10 years does not reveal the presence of structural and bony changes as the prevailing cause of low back pain. These cases were seen not in an orthopedic but in a consulting industrial practice where the conditions are quite diversified, the industrial cases coming mainly from one large plant employing from 1000 to 1500 men, often doing the fastest and heaviest kind of work. During the entire period it was not necessary to treat the many hundred back cases by extension or by orthopedic surgery. No final total disability claims were ever established in any of the so-called lumbosacral or sacro-iliac injuries treated by us during this 10-year period.

The Eucupin Solution in Oil injection treatment was the logical results of my hypothesis concerning the etiology of most cases of low back pain. It proved to be a very valuable aid in the treatment of these conditions.

"X-ray Positive" back cases (fractures, dislocations, severe structural bony changes and defects) require competent orthopedic treatment. Oil injection may be advantageously used for symptomatic relief of pain.

"X-ray Variable" back cases (cases showing x-ray variations from the ideal normal, as encountered in the lumbosacral and sacro-iliac region), often require no specialized orthopedic treatment. These cases always should be treated conservatively at first and oil injections will remove pain and hasten recovery.

"X-ray Negative" back cases (strains, sprains, bruises, inflammation, referred back pain, statural, postural and functional backs) should be treated conservatively. Injections with Eucupin Solution in oil will remove back pain and hasten recovery.

Low back pain and sciatica may originate in the lumbosacral nerves and their branches. A certain tender area does not establish it as the point of origin of the pain, for low back pain and sciatica are often referred. Inflammatory irritation of the lumbosacral nerves is a frequent cause of low back pain and sciatica.

Low back pain is often caused by irritation of the posterior nerve branches and may be successfully treated with Eucupin Solution in Oil injections.

Sciatica is often caused by irritation of the anterior nerve branches and after conservative treatment has been tried, may, in most cases, be successfully treated by perineural injection of 0.1% B-Eucaine in normal saline solution around the sciatic nerve.

The most desirable point for oil injection was found to be beneath the transverse process of the 5th lumbar vertebra, which is found by the intersection of anatomical lines described in the technique. The prolonged analgesic action of the Eucupin Solution in Oil not only affects the muscles, fasciae, and nerves locally, but also apparently is conducted towards the nerve centers.

Eucupin Solution in iodized oil injections proved to be very successful in the treatment of myositis and neuritis in various parts of the body, other than the back, a cure being effected in such a condition as torticollis.

Where diffuse myo-fascitis is caused by massive inflammation the oil injection treatment is much less effective. This type of case requires much larger quantities of anesthetic oil and the injections have to be made into various areas, and frequently have to be repeated at an early date.

Where referred pain is caused by pelvic disorders in the female, oil injections give good results. However, in some cases the back pain recurred after two to four weeks and reinjection was necessary.

Eucupin Solution in 20% iodized oil was developed primarily for diagnostic purposes, but our observations reveal it to have even greater therapeutic possibilities than the plain oil. Eucupin Solution in 2% iodized oil possesses these attributes but is not opaque to the roentgen ray and will not, therefore, interefere with later visualization of the part.

While this oil injection treatment has proved to be a valuable, new therapeutic procedure, it should not be considered a panacea for all cases of low back pain.

A careful true history is most important in every case, together with a thorough physical and x-ray examination. Only after careful search for the causes of the back pain should the Eucupin Solution in iodized oil be used. If this routine is carefully followed the immediate relief obtained will not obscure the presence of pathological conditions which may require more radical orthopedic procedures.

[I am obligated to Dr. James M. Nelson for the compiliation of statistical data, to Dr. David S. Dann and Miss Lora Robins for radiographic and photographic assistance, and to Rare Chemicals, Inc., Nepera Park, New York, for their cooperation in providing Eucupin Solution in Oil and Eucupin Solution in iodized oil.]

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Some Wired Fractures

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FROM time to time different methods have been tried to hold the various fractures in proper position, but I will not undertake to describe them, confining my discussion to the use of wiring:*

(1) J.P.F., a brakeman, was injured in 1906 by falling from a box car. He sustained a transverse fracture through the middle of the thigh. Some kind of extension, either Buck's or Hodgen's splint, had been used on him by a surgeon in another city, but when he reported to me several months later, the fractured ends of the bone were overlapping, he had about two inches shortening and non-union. We decided to do an open operation and wire the ends together with silver wire, which was the only kind of wire we knew at that time for this kind of work.

An incision about six inches long was made on the outer and posterior side of the thigh over the seat of the fracture. The leg was carried across the other at a right angle, which enabled us to bring both ends of the fracture through the opening. There is not much soft tissue between the skin and the bone at this location. About an inch was sawed from the end of either bone and holes were drilled entirely through each, parallel to each other. The largest silver wire in use at that time was passed through one of the bones and brought back through the other and the ends twisted together. Just as I was making the last turn it broke and I had to insert another wire. This had been the rule and not the exception with me, and it had happened many times. A plaster cast was then applied, extending from the foot encasing the fractured limb, the abdomen, and the chest up to the axillae. Union occurred at the usual time and with the result shown in the picture which was taken recently. The fracture gave him no further trouble except the shortening, and he has been working continuously since it united. The silver wire has become disintegrated and scattered through the bone, but it held long enough to serve its purpose. Whether this disintegration was caused by chemical action or cell proliferation I do not know. I once saw a surgeon operate for a similar condition, but he made his incision down through the quadriceps muscle, and it was a much more difficult operation.

A short time after I operated on this case I bought some stove wire and had it silver-plated so as to give the silver contact with the tissues. This wire has the strength to hold the bones in place and will not break when you twist it up as close as you choose. I have been using this wire ever since, but recently a rustless wire has been put on the market and is preferable where you need only a very small wire. However, there are many conditions where the larger wire is preferable.

If I had a patient like this one to treat now, I would remove very little of the ends of the bones using a bone curette or forceps, merely freshening them up as it were, before introducing the *silver-plated* wire. I would also put in two wires, one laterally and the other at right angles to it after boring the holes in the bones about three-quarters of an inch from the ends. The tissues in this location will tolerate this amount of hardware, and the ends could more easily be held in place. I would also use the plaster splints in preference to the cast. One splint reinforced by a metal strip at the place over the fracture should be extended from the inner malleolus under the foot up the outside of the leg and thigh to the axilla and another from the crotch down and overlapping the former at the sole of the foot.

* Presented at the 1938 Annual Meeting of the Association of Surgeons of the Southern Railway. (The Figures refer to the Cases as similarly numbered.)

Fig. 1.

Fig. 2.

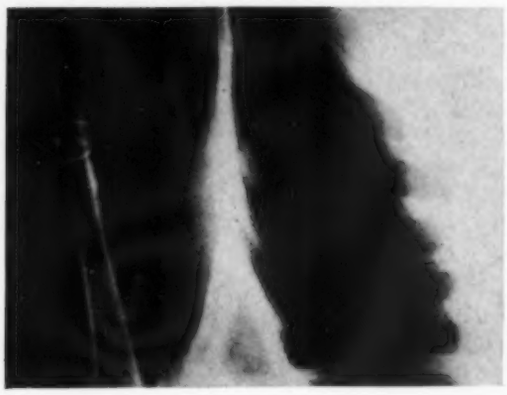

Fig. 3.

The first should be bandaged with a roller bandage from the foot up to the crotch and ending in a spica bandage. The upper end of the long splint should be held in place by a wide strip of adhesive which should be carried entirely around the chest near the axillae. The inner splint should then be put in place and bandaged from the foot upwards.

I prefer the splints to the cast because when the muscles begin to atrophy, the bandages may be removed from time to time and applied so as to make the splints fit snugly. This method would leave very little shortening, less than an inch probably. Lane's plates were once recommended in these cases, but were not satisfactory for several reasons: the screws would not hold in the sides of the bone, the tissues would not tolerate so much metal and practically all of them had to be removed sooner or later. I have not had an occasion to remove a wire from bones which were put in closed fractures, but usually have to remove those put in open fractures as the wound is infected before they are put in and we have no way to sterilize them thoroughly. The sterile bone-pegs inserted in the medullary canal have been satisfactory in the hands of some operators, but I would rather depend on the wiring.

(2) C.E.S. This is a case of a fracture of the middle of the femur which has been recently wired properly after traction had failed to hold the ends in position. The muscles are very strong in the thighs and it requires a plaster cast or closely applied splints to hold them in place after they have been wired.

(3) J.R.L. A special delivery messenger was injured September 7, 1936, by being thrown from his motor cycle. This fracture was about the middle of the left forearm. Soft tissue caught between the ends of the bone so that I was unable to get the ends together by manipulation. An open operation was done, the incision being made on the outside of the arm where very little soft tissue is met before reaching the bone and none of the important arteries or nerves is located. The scar at the line of incision is barely noticeable and the function of the member is perfect. This condition could never have been obtained without the open operation.

(4) E.C., a mail carrier, was injured August 15, 1935, when his mail car collided with another automobile and turned over, catching his elbow. The lower end of the humerus was comminuted, and his fellow workers were satisfied that he was through carrying the mail. An open operation was done and the fragments wired together with the result shown in the picture. There is no deformity and the function of the limb is perfect.

(5) J.C.M., a special delivery messenger, was injured September 24, 1935, when his wheel collided with an automobile. He had a dislocation of the outer end of his left clavicle. I believe that this condition can only be corrected by wiring, which is a simple operation, if done under a general anesthetic. I have done several under local anesthesia, but all of them complained when the holes were being drilled in the bones. The small rustless wire is the best in these cases. It should be passed through the acromion process and up through the under surface of the clavicle. The articular surfices are put in place and the wire twisted so as to hold them, and the ends tucked under out of the way.

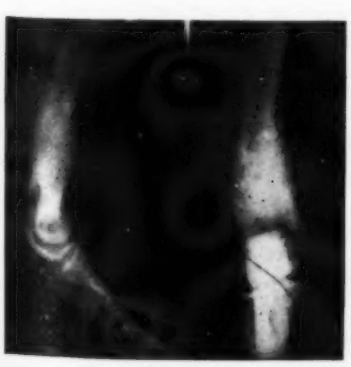

Fig. 4.

Fig. 5.

Fig. 6.

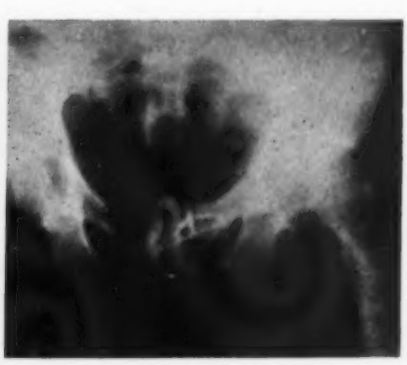

Fig. 7.

This boy has no deformity and no loss of function. This dislocation does not occur very often and is usually produced by being rolled between two vehicles or cars.

(6) V.P., a colored switchman was injured when we were still using the silver wire. Both bones of his right forearm were broken and we could not hold them in place with splints. It is not pleasant to report failures, but this accident happened when we were making experiments, blazing the way as it were. I had forgotten the case till he reported to me recently for examination for the Railroad Retirement Board. He has non-union and there are many things that he can not do with this member. The picture shows that the wire has been broken in several places, and it seems that the wire was passed through only one layer of either bone. If a silver-plated wire or one of rustless steel had been passed entirely through the bones and twisted properly, the result would undoubtedly have been far different. This was a poor job and I might have been justly criticised for it, if any one else at that time had known how it should have been done. Occasionally we can get by with poor work and we may be criticised for the best work that could have been done under the circumstances.

(7) M.H.B., a switchman was injured July 10, 1930, by being rolled between two box cars, suffering separation of the pubic bones and a sacro-iliac luxation (complete). A general anesthetic was given and the dislocation of the sacro-iliac joint was reduced while he was lying on a cushion on the floor. An attendant held both hips down on the cushion and the lower limb was rocked back and forth with the knee held perfectly stiff. Then he was placed on an operating table and the pubic bones wired together. The silver-plated wire will stay with him as long as he lives. It seems to be placed towards one side, but it was placed in the middle originally and this appearance may be a fault of the picture. This was put down as a permanent disability and settlement was made on that basis. He has been farming and gets around fairly well, but evidently he could not do the work of a switchman as he could not climb about on box cars.

I WANTED to show a wired patella, but I can not locate a case at the present time. It is the ideal treatment for these fractures, especially when they are comminuted. They can be done painlessly under local anesthesia, but I prefer a general anesthetic unless it is contra-indicated. A crescent-shaped flap is made and turned up over the patella, and clotted blood or small fragments of bone are removed; then a small wire is passed through the patella tendon, through the fascia at the side of the patella, then at the top of the patella through the quadriceps tendon, and again through the fascia at the opposite side of the patella. The fragments are placed in position and the wire is drawn together, and twisted, and the ends put under where they will not be irritating, and the wound closed. Either a cast or plaster splint applied on the posterior aspect of the limb from the heel to the pelvis may be used. The kangaroo tendon has been found to be unsatisfactory, as the knot may work out long after the patient is supposed to be well. When I commenced to study medicine a fracture of a knee cap was considered a permanent disability.

Fractures of the neck of the femur are being treated by passing a steel pin so as to hold the head and neck in position. This is only a modification of the operation done in New Orleans years ago by PROF. EDMUND DENEGRE MARTIN, who used a long screw in place of the pin, and it may be a better way.

Silicosis—Case Reports

DR. M. CONROZIER,
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and
DR. J. MAGNIN,
Ex-Interne des Hopitaux de St-Etienne, France

WHEN this subject was proposed to me I must say that I felt some astonishment; I expected that they would ask me to entertain you on silicosis and they told me: Don't touch it.* And I thought that it was one of the questions in France as well as in Belgium upon which it was advisable to think, but prudent not to talk too much." (PROFESSOR LANGELEZ: *Le Medecin d'usine.* — Conference held May 17, 1938, in the *Journal for Studies of Occupational Diseases and Industrial Hygiene,* organized by the Union of Metallurgical and Mineral Industries.)

Such are the words recently uttered by the Belgian savant at the beginning of a conference upon "Some Professional Intoxications Due to Metals." Perhaps we should impart this opinion, which is given with a certain mystery. Our naivete goes beyond this and, as last year, we now render an account of the state of silicosis in our mining basin.

We may have thought that he had exhausted our list of cases, but this is by no means so. We have seen and checked by radiography 42 new cases. If one imagines that the Gard Basin is of modest importance, he cannot fail to be impressed with this figure. It extends, to 130, the number of victims seen and radiographed. We knew, however, that a considerable number had escaped us. In random questioning of miners whose observations we present here, we took the names of fellow-workers deceased "with the rock malady" during recent years who were unknown to us. And these cases have been numerous.

There is no doubt that difficulties of exploration are the cause — and the only one — of the apparent rarity of silicosis in certain mines. We know, after an experience of nearly 10 years, that one only finds silicosis, indeed, when he investigates with-

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* From: *La Medecine du Travail,* 10,5:173-248 (September) 1938; Translated by EMERY R. HAYHURST, M.D. Only excerpts are rendered herewith from this extensive article, which is likewise illustrated by 29 half- and full-page half-tones of chest roentgenograms. Through the courtesy of DR. CONROZIER, three of the illustrations are reproduced herewith, being those especially selected and legended by him for publication in INDUSTRIAL MEDICINE.

out restraint. We complete our thought by adding that everywhere in France one can find numerous cases of silicosis — if he really wishes to. Two recent examples illustrate this opinion excellently. The coal mine basin of Saint-Etienne shows this amply in regard to fibroses studied in miners which have been portrayed in a recent work of our confreres. We know nothing about the publication of cases of silicosis observed in the gold mine at St.-Pierre de Montelimar. But, we have seen in the last year three magnificent cases of nodular silicosis in the development of this mine, and, if we can believe these afflicted workers, there is no reason why these should not be particularly marked. They have it, as they say, because they have seen many of their comrades die of it. This information came to hand because these survivors — of whom one died of another cause — were stranded in our basin, so that one came to know that, in the gold mine at Anjou, silicosis exists and has its victims. What we have learned of St-Etienne and of the gold mine, and what we know about the coal mines of the North and, equally, of the iron mines of the East, causes us to conclude that silicosis exists everywhere that rock is drilled (perforated). One finds it when he wants to look for it. The reasons for its apparent absence in certain regions have nothing in common with real scientific research.

The figure of 130 victims merits our attention for a moment.

We have repeatedly insisted upon the enormous amount of sickness which accompanies — in our basin — work upon rock. In fact, a malady so widespread with us that we have observed 130 cases within a few years. At the same time numerous others escaped us. Thus we are not concerned with doubtful cases, as all the cases described by us under the term "silicosis" are mature lesions — nodular or tumoral.

Of our 130 cases we possess nearly 300 radiographs. Certain of these cases have been followed for nine years. Most of them have been seen at various times with the screen. All, with few exceptions, have had repeated bacteriological controls. As, for the most part, the average of our cases appears richer in lesions of fibrosis than in other regions — in the North in particular — we can, we believe, speak of silicosis as a malady familiar to us and one which, we believe, we can easily recognize.

Respecting Our Own Cases

NINE years have passed since the first case came to our notice. During these nine years we have written extensively upon the subject of silicosis. We think, however, that we have not portrayed our cases vividly enough to our readers. It is necessary to see and to understand these cases. We essay to publish our findings about them this year.

[There is next presented the details of 24 cases for which illustrations are shown and another case which is briefly referred to but without an illustration. The following discussion concerns, first, *nodular cases*, then *mixed lesions* of nodular and tumoral image types, then the essential characteristics of *other silicotics*, including *pure tumoral image forms*, and terminates with a discussion of *tuberculosis complications*, — E.R.H.]

A Typical Nodular Case

Among our "nodulars" we desire to present detailed observations of St. (p. 183 and Fig. 1, in the original paper, and Fig. 1 herewith). This case is a very beautiful one. This man of 34 years, who worked three years drilling

No. 1 St. 34 years of age. Three years at rock work between 1930 and 1936. Still at work one year after this radiograph. A very vigorous subject. Investigation for Bacillus of Koch negative. A typical case of nodular silicosis

(perforation), was still working on the rock when the first radiograph was made, and we know that he was working there recently. His general state of health was excellent. Bacilloscopy. Dyspnea. The contrast between the splendid vigor of this man and the lesions which show in the film is typical.

Tuberculosis Complications

(The authors are careful to state, p. 200):

"We have not taken for a pure silicotic a tuberculous triviality abnormally resistant to his lesions. Our reports concern, in reality, only type-observations of silicosis seen in our basin."

(The series terminates with the presentation of cases of *three tuberculous miners* seen in the last year):

Jak. (p. 200 and Fig. 17 in the original paper — this Figure is not reproduced here), aged 40, worked 14 consecutive years exposed to dry rock dust in the same mine — from 1922 to 1937, and was still working when x-rayed. The supervention of tuberculosis hastily modified the development of his case. He entered the hospital. The entire left lung and the lower half of the right lung presented a picture of pure nodular silicosis. Only the right summit suggested a massive lobular infiltration of irregular denseness, containing a probable cavity.

It would seem to us — after having seen the same in other cases — that the history of pneumoconiosis in Jak. may be summarized thus: He had, during

his last year of work on the rock, gradually developed silicotic lesions, but 14 years of alveolar traumatism created local conditions favorable to secondary tuberculosis which recently became noticeable. It is remarkable, in fact, that the two types of lesions, tuberculosis and silicosis, are only superimposed in the zone at the right summit; everywhere else the lesions are pure silicosis, as the x-ray picture shows. Here was a miner who came to consult us only a few months before his death.

CAS. (p. 209 and Fig. 11 in the original paper — this Figure is not reproduced here), age 45, came to see us this winter because he was in a state of chronic asphyxia. He had been eight years on the rock, between 1925 and 1936. His dyspnea had become practically arrested. When he came to see us, he was in full physical vigor. We examined him only a few days before his death. He was cyanotic, complained of pain, but retained his full muscular state. We did not have the opportunity of examining his sputum. We declare, in other respects, that this examination, which we customarily carry out in a systematic manner, was of secondary interest.

All of the observations which we have made on our cases in the last 10 years convince us that tuberculosis develops very rarely with us, and nearly always tardily. We shall discuss fully, later on, the question of silicosis and tuberculosis, but the easiest way of comprehending these reports is to dwell here upon our four years' observations. This concerns very dissimilar cases, but which, precisely by their diversity, show the peculiar aspects of the malady and better place the question upon sound ground.

K. (p. 209 and Figs. 22 and 23 in the original paper — these Figures are not reproduced here). We showed an x-ray picture of this case last year. Aged 37, worked on the rock for six years between 1928 and 1936. His comrades vouchsafed that he worked without regard to precautions to his resistance. He was, in fact, very vigorous when he was seen by one of us for the first time in August, 1936, but since April, 1937, has been forced to quit work entirely. He next became incapable of any effort. His first x-ray indicated very prominent lesions. The second, obtained some months later, showed a very acute extension of the lesions.

At no time did K. show the least suspicion upon auscultation, in which only silence and areas of blowing sounds alternated. Multiple tests were made on this man for the Bacillus of Koch — five direct examinations, five homogenations, and three guinea pig inoculations. All the tests were negative.

In contrast to K's case that of TAL. is presented (p. 210 and Figs. 24 and 25 in the original paper — these Figures are not reproduced here). Whereas K's case developed rapidly, this one advanced without other impairment than gradually developing dyspnea along with lesions becoming gradually more prominent. As with a lot of our cases, TAL. was discovered accidentally. The chief features are: he fractured his vertebral column in the course of an industrial accident. At that time an x-ray picture was made which provided the first picture. That was nine years ago. This picture, taken under difficult conditions, showed definite silicotic nodulation. His grave injury and total invalidity condemned him to complete rest and placed his silicosis under the best conditions for treatment. Furthermore, it combated any tuberculosis activity, but, in 1938, TAL's thoracic picture was as shown in Fig. 25. The lesions have progressed and have conglomerated at many points.

Have his general condition and local signs followed a parallel development?

Entirely to the contrary. TAL. has splendid health. He weighs 84 kgs. and his only complaint is that of easy breathlessness. We thus see, after nine years, that the lesions have progressed in the chest of a former miner — extensive bilateral lesions — without any alteration of his general state of well-being. These lesions act in his case exactly like foreign bodies. They play a purely mechanical part: TAL. has been breathless for a dozen years.

SKR. is a case — a rare one for such a degree — of massive silicosis with rapid development (p. 210 and Figs. 26 and 27 in the original paper — figure 26 is reproduced herewith). This very young man (34 years), built like a Hercules, developed, within a few months under our eyes, an extremely rapid evolution of a true tumoral silicosis, appearing in large blocks.

We first saw this case November last. It has been our most beautiful case of massive silicosis, for everything is shown.

No. 26 Skr. 34 years of age. A very vigorous subject. Four years on the rock between 1933 and 1937 and continuing such work since this radiograph was made. A typical case of massive silicosis without nodules. Rapid asphyxial evolution. Absence of Bacillus of Koch

This man's work on dry rock commenced a short time before, in 1932, but was carried on strenuously by a worker with the musculature of a wrestler who could work long days; absence of all auscultatory signs other than areas of silence; typical radiographic picture; a massive fibrous block in the midst of each chest; two microscopic and two inoculation investigations negative.

Eh! Well, this man, who was working on the rock at the time of the first radiograph died from suffocation within a few months. We saw him before his departure for his native Poland to which he returned, as he said, to see his own people again before dying and, in fact, he died on the 15th day after his arrival. His last picture, taken on the eve of his departure, showed a quasi-total invasion of the chest; and an enormous block in each hemithorax. His last inoculation in guinea pigs was made at the same time and we knew the result after receiving the news of his death.

We acknowledge to those who hold to the tuberculosis thesis that we were not able to get an autopsy on this man. But, we know well that it was the dust which killed him.

Pneumothorax in Silicotics

NEXT we present our last case. It is worthy of very important comment for it especially raises the question of distinguishing between silicosis and tuberculosis.

Pr. (p. 219 and Figs. 28, 29 and 30 in the original paper. Figure 30 is reproduced herewith). This concerns another young man: 36 years. He did not get silicosis in our basin, but in a gold mine at Anjou where he worked seven consecutive years, from 1930 to 1937. He did not come to our attention until he had been with us a month on the rock. In July, 1937, his general state was still good, but Pr. showed fatigue and was dyspneic and coughed. Nevertheless, he continued to work in the depth to November when he was examined for the first time. He had had, during preceding days, a stitch in his side without thoracic drama. We were very surprised to discover a large left-sided pneumothorax.

No. 30 Pr. 36 years of age. Seven years at work on the rock in a gold mine. Double pneumothorax without fever, asphyxia, or Bacillus of Koch. The man was still living eight months after this radiograph was made

Some weeks after that, we had a new surprise: the right side showed, in its turn, a complete pneumothorax at which time the left had resorbed its air. We regret, in passing, that material difficulties prevented us from making a new picture at the moment of complete collapse.

In the beginning of 1938 — a new aspect: the left pneumothorax recurred with complete collapse of the lung, while the right pneumothorax was nearly completely absorbed.

Finally, in the springtime, the right side showed a very cleancut picture of pneumothorax.

There is no reason, henceforth, why this game of see-saw should not continue. The man resisted rather well these repeated pulmonary episodes. At no time did he show a febrile upshoot. At no time was there any evidence of effusion. Two bacteriological examinations with homogenation and two guinea pig inoculations were negative.

We hesitate at this time to dwell at length upon a case which appears similar to that published last year by M. Roubier. But the great interest it creates merits, meanwhile, some reflections now.

Here we have a man with both lungs crammed with confluent nodules who shows one of the most dreaded complications of pulmonary tuberculosis: pneumothorax. It has not limited itself to one site: it has selected two places and further complicates the situation by repeating on each side.

We have had considerable experience with pulmonary tuberculosis. We have, likewise, a long familiarity with therapeutic pneumothorax. We know how grave the survival is in pneumothorax in the case of ordinary tuberculosis and we know the lamentable end is nearly always spontaneous pyo-pneumothorax.

We dread no less pulmonary perforation in our operations of artificial pneumothorax. We have lost, up to this time, all our cases having perforation into the general pleural cavity. Now appears a man in whom both lungs are riddled from top to bottom, an undoubted case of tuberculosis, according to MM. Rist and Policard; yet, a man, we say, who shows double spontaneous pneumothorax without any evidence of temperature elevation, without any effusion, and without expectorating any bacilli.

In what other affection than silicosis has this been seen — we earnestly ask? Where is there a parallel pathologic curiosity? We have now discovered, in our clientele, three cases of spontaneous pneumothorax in subjects apparently non-tuberculous, who have evolved, without complications, to resorption of the air. However, in each case, the lungs had uncomplicated lesions. Comparing what appears comparable, we come back to the discussion of true tuberculosis, and we say that they have never, to our knowledge, presented a picture comparable to that of Pr.

It is probable that medical literature has recorded (we do not know it) cases of spontaneous double pneumothorax in bilateral tuberculosis. We affirm without hesitation that the survival of the victim was only a question of days; that a liquid pleural reaction appeared immediately; and that the Bacilli of Koch swarmed in the expectoration.

We are cognizant of what may be said:

"These cases of latent pneumothorax among your rock workers can be explained by the peculiar constitution of the nodule appearing in the given case: silicotic fibrosis is anatomically of such a nature that it encompasses the active tuberculous element and permits the rupture of the nodule in the pleural sac without any scattering over the serous membrane."

This is a clever explanation! Thus, it is natural that workers on the rock are the only instances of apyretic and aseptic pneumothorax. Such is one of the effects of silica.

It is not astonishing that arguers who deny the mal-effects of silica when it produces lesions of fibrosis in miners may be precisely the same ones who attribute an important role to it otherwise. Assume that the pneumothorax is aseptic? This is because of the silica. Assume that the sputum does not contain bacilli? This is because of the silica.

But assume that, perchance, silica were the

cause, the unique cause of the malady? Then tuberculosis in the rock miners would be easily explained: The widely spread bilateral lesions (one respires, is it not true, with both lungs), its indifferent evolution, the paucity of local signs, the mechanical character of the dyspnea, cause enormous lesions which in no way influence the general state of health, and comport themselves as foreign bodies. . . .

Then, finally, a spontaneous pneumothorax, which is grave! This puts a tuberculous focus into communication with the pleural cavity and one knows the sensitivity of the serous membrane! A nodule which bursts into the pleural cavity opens up within this cavity not only a siliceous hull which, they say, protects, but, fatally (to the argument—E.R.H.) also the identical substance of the nodule which, to them, is a bacillary lesion in a state of activity. For it is indeed necessary, is it not, that pulmonary tuberculosis take some part, for instance, in the production of tubercles? And as the pleural cavity fills with air, it is necessary that this air traverse the very small tuberculous focus of the nodule before it spreads out in this closed space. And this air, which likewise fills the serous cavity, rushes in from the bronchi and bronchioles opening up everything within the bacillary foci, for PR., we do not doubt, is crammed with nodules from the apex to the base of his lungs and in every part of the parenchyma. Can we say that the pleura should remain aseptic on both sides, and should absorb its air and replace the same again with impunity? Can we say that the victim, among the local miners, would be a tuberculous individual so far as the lungs go, but a silicosis individual so far as the pleura goes? Considering all the arguments, as we see it, they reduce to absurdity.

We now return forthwith to clinical facts. Let us adhere to the truth. It isn't the tuberculosis which counts: It is the siliceous dusts and, since we are upon the subject of pneumothorax, we do not refrain from saying a few words concerning an unpublished case of artificial pneumothorax in the instance of a victim afflicted with silico-tuberculosis.

We have kept under radiographic control, from the beginning of last year, the chest of a rock miner who had silicosis of the nodular type, but complicated at the right summit with very widespread shadows plus a "bread-crumb" aspect indicating tuberculization. This case expectorated bacilli continuously. He had to quit work and developed as a case of tuberculosis: hemoptysis, fever, cough, expectoration, emaciation — the left lung riddled from apex to base with nodular shadows.

Eh! Well, in spite of this state of the left lung we did not hesitate to produce on the right side — the side with the bacillary lesions — an artificial pneumothorax.

The result was immediately excellent: The man increased his weight in a few months by 12 kilos. He developed, unfortunately, later on, a premature coalescence and now the lesions on the right have become reestablished.

It is nonetheless true that we were able to undertake an artificial pneumothorax in a case affected with bilaterally spread lesions. We were able to do it — and the immediate success confirmed our convictions — because the lesions of the left lung and the inferior part of the right lung were lesions of pure silicosis. We would have judged insane — and believe all phthisiologists would agree — the performance of a right pneumothorax if the lesions on the left, in the case, had been those of tuberculosis. They were so insignificantly such that we undertook an extra-pleural pneumothorax against the lesions of the right summit.

Some Observations Apropos of Our Cases

WE COULD prolong the presentation of our cases because we have numerous living cases in our files. They are exactly similar to those which we have summarized.

Through experience and period of observations, we have enriched ourselves with some knowledge of this malady. We simply report the essentials briefly, directing our whole attention to the problem of tuberculosis — which is the crucial point of discussion.

Has the Duration of Work on the Rock Any Bearing upon the Gravity of the Lesions?

ALL THINGS being equal, such is incontestable, and the logic is also. We have recounted, above, observations upon miners who, in spite of dyspnea, have prolonged their period of work upon the rock; their afflictions have progressed rapidly. Ordinarily more than four years are necessary to produce silicosis. How comes it then that we have cases of silicosis developing in less than four years and in one case after 18 months of work on the rock?

There are several reasons. For example, the cases of rapid development are, for the most part, cases where the work was dry and under very dangerous conditions. The vertical depth, which amounts at best to a bath in a tub of dust, is the origin of most of our precocious silicosis cases. The work, which was manual in times past, was on the contrary, only slightly dangerous and required considerable time to produce silicosis.

Another reason: Sometimes miners, from their own trustworthy statements or those of their comrades, thoughtlessly breathe deeply, and with wide-open mouths, thick gray clouds of finely disseminated dust. The cases of KOU. (p. 209 and Figs. 22 and 23 in the original paper — not reproduced here) and SKR. (p. 210 and Figs. 26 and 27 in the original paper — Fig. 26 is reproduced herewith) are examples.

Other miners are prudent by nature and breathe with utmost care, using their noses as much as possible and sometimes wearing a moist sponge in front of the mouth and nose. These are the ones who seem not to have furnished any of our precocious cases. The injection of water at the drill point has certainly delayed the beginning of silicosis.

All said, there remains, nonetheless, the fact that duration of work on the rock has often no

bearing on the gravity of the lesions. We published last year the case of N. (not illustrated in the original paper) whom we have followed for three years: this miner, who submitted to sanatorium care, from which he strayed and then came to us, after having very numerous negative examinations of his sputum (a score!) which never killed guinea pigs, developed a massive silicosis with enormous shadows after 18 months on the rock.

At the other end of the scale, we know a case which, after 16 years in dry work, had only insignificant fibrous aborizations (he finally had to quit because of supervening dyspnea).

Between these extremes may be observed the greatest variety of cases where the intensity of the lesions has shown no relation to the duration of exposure to dust.

Will not tuberculosis explain the difference? One should admit the reality of pure silicosis in certain cases, but associated with tuberculosis in other cases. For these latter, who are rare in our basin, there is no doubt that the appearance of tuberculosis cuts short the evolution of the case. But we are discussing here that phase of the question which concerns the influence of duration of work on the rock upon the malady produced. Further, this duration, which accounts for precocious or delayed appearance of silicosis, equally accounts for its evolution, that is, the defense of the upper respiratory tract, which is the naso-tracheal coefficient of protection against dust.

We again refer to the case of N., who developed silicosis after 18 months on the rock, and of P., who failed to develop it after 16 years. N. is one who shows enormous lesions in a robust man of good color. P., on the contrary, is a small man of stunted development.

No, it is not tuberculosis which calls forth the appearance of precocious lesions but the density of the dust, its high content in silica, the carelessness of the worker and, finally and especially, the state of the respiratory passage-ways — the upper respiratory tract especially, but probably also the condition of the respiratory tract in the interior of the lungs.

Does the Nature of the Dust Determine the Anatomic Form Which Silicosis Takes?

ONLY having experience with the dust in our basin, we cannot generalize our conclusions. We can say, so far as our observations here go, that, with the same type of dust, the whole gamut of radiographic aspects of silicosis occurs. We state precisely, as all observers, that we more often see the nodular forms (separate nodules or conglomerations in pseudo-tumorous masses), and, less often, massive forms. Thus, our three gold miners were nodular silicotics.

It is indeed certain, on the other hand, that it is not without import what dust can cause silicosis. It does not suffice that silica be present. There must be a great deal of silica. And, it is probable that, for an equal quantity of silica, certain rocks produce, under the drill, very deadly dusts. There occurs without doubt a crystallographic state of the pulverulent element. This is more or less deadly according to its form, its roughness, and its injurious properties. But it is not solely a question of the quality of the dust. The anatomic form of silicosis depends at least in part on the manner in which the dust affects the alveoli.

We were, we believe, the first to point out *(Journal de Radiologie d'Octobre 1937)* this essential idea: That pure tumoral images — those which form in dense clear-cut blocks, which the radiograph shows resemble sarcomatous metastases much more than bacillary lesions—that these images, we repeat, are most often found isolated in a parenchyma apparently intact or where nodules are rare or absent. We have concluded that, very probably, these tumors have no causal relation with the silicotic nodule. The nodule is not therefore always the beginning form of silicosis. There are anodular silicotic tumors and, for these, the best explanation would appear to be lymphatic blockage suggested by American authors. To produce this blockage, it is necessary that the dust arrive at the pulmonary alveoli in such density that the means of elimination and of defense (dust cells) be out-flanked. The accumulation of siliceous dust *in situ* would be, in this case, the basis of the malady. All pulmonary territory being thus invaded, fibrosis intervenes by transforming *en bloc*, and densely, the parenchyma concerned. One can thus explain how fibrous blocks of silicosis form in the most used region of the lungs, where the air arrives easiest, and where the dust consequently arrives with the greatest density and speed. One can explain thus, equally well, the remarkable symmetry of the lesions. Finally, the personal coefficient of protection of the upper respiratory tract against the dust here plays again an important role.

Is the Evolution of Silicosis Controlled by Precise and Easily Revealable Factors?

WE CANNOT misguide ourselves in digressions in which the actual state of our knowledge does not permit drawing useful conclusions. Certain lesions similar in appearance evolve rapidly, others very slowly.

Del. (Fig. 3 — this Figure is not reproduced here), who worked 10 years without interruption, came abruptly to be a respiratory invalid, Ch. (Fig. 6 — not reproduced here), on the contrary, scarcely developed after 20 years. Others like N. were silicotic within a very short time and then developed no further. Du., who has enormous lesions, and followed by us for over 10 years, developed only slowly. Finally, others like Skr. (Figs. 26 and 27—Fig. 26 is reproduced herewith) developed a sort of galloping silicosis, after a short sojourn in the dust.

What factors intervened then in the one case to precipitate development, and, in the other, to permit prolonged survival?

We think that in the development as well as in the initiation of silicosis, and equally in the anatomic forms of the malady, two principal factors

present themselves: (1) The imprudence of the worker who, working at the task, breathes without precaution and, more especially, deeply as he endeavors to earn more; and (2) the individual coefficient of protection.

We do not think that resistance to silicosis should be linked closely with physical vigor. SKR. (described above) was a Hercules. CH (described above) was a weakling.

Tuberculosis does not explain the differences in development. On the whole, the cases in which the lungs show the most marked lesions are not those which expectorate bacilli.

ARE THE PURE MASSIVE FORMS TOLERATED BETTER THAN THE NODULAR FORMS?

THIS is the opinion of prominent authors. It is also in part our opinion. We have already spoken of miners who carried their fibrous masses like foreign bodies. But we have not forgotten that certain of our early nodular cases are still living.

The dramatic thing about these cases is to know whether they are doomed or whether they can survive at the moment we discover the malady. We have previously published the case of a miner who protested vigorously when we interdicted his working upon the rock because he had only discrete silicosis and who died in misery three years afterwards, from irreparable extension of his lesions.

(A resume of the authors' views upon Silicosis and Tuberculosis completes the article—pp. 237-248; not included in the foregoing translation—E.R.H.)

New Books

Review by

FREDERICK W. SLOBE, M.D., F.A.C.S.

HERNIA, by Leigh F. Watson, M.D., (published by C. V. Mosby Company, St. Louis). This is the second edition of Watson's well-known book containing several additions and changes, notably a very comprehensive section on the injection method. It is always a pleasure to read Watson on Hernia as he has been a close student of the problem for years and a prolific writer on the subject.

An interesting historical sketch is presented, the history of progress in the treatment of hernia following quite closely that of surgery in general. Surgery is traced from the Sumerians in 4000 B. C. followed by the Babylonians, Egyptions, and Greeks, Hippocrates exemplifying the latter. The Roman Celsus in the early A. D.'s frequently operated for hernia. Galen, the famous Greek, in the second century ligated the sac near the external ring. Rhazes exemplifies the progress made by the Arabs in the ninth century and in the eleventh century the Europeans took the leadership with great progress being made during the Renaissance. Lavater's book in 1619 was one of the earliest writings urging operative cure for strangulated hernia, and the dawn of the modern era was said to have begun with Cooper and Scarpa in the early nineteenth century.

Some fairly comprehensive chapters are devoted to anatomy, etiology, symptoms and diagnosis, complications, partial enterocele, hernial tuberculosis, omental hernias, and fatty hernias.

The author agrees with the quite generally accepted opinion that in the etiology of indirect hernia the congenital performed sac is the essential factor. There is, however, a discrepancy noted on page 125 where the author states that, "given an exciting cause that increases intra-abdominal tension, the preformed sac together with a knuckle of small intestine or piece of omentum is forced through the internal ring," whereas on page 136 he stresses the accepted prevailing opinion as follows: "congenital hernias are due to the persistence of a patent processus vaginalis, and a sudden strain or trauma merely forces the abdominal viscera into a congenital preformed sac." Murray is quoted as having found 21 empty sacs in 100 postmortem examinations of individuals who had exhibited no hernia during life.

In the differential diagnosis between direct and indirect hernia the frequent impossibility of accurately distinguishing between the two pre-operatively is not emphasized.

In the operative treatment the author has purposely not discussed most of the operations except those which he prefers. The following operations are described and well illustrated: Marcy's method in 1881, the modified Bassini in 1883, the modified Halsted method in 1903, and the Andrews method in 1895. More briefly are the methods of Stetten and Scott whose modified Bassini procedure appears to be the method preferred by the author.

In the operative treatment of direct hernias the methods of Bloodgood, Blake, Davis, Hoguet, and Downes are described, the latter being preferred by Watson. Operations using fascia lata as described by Kirschner, Gallie, Le Mesurier, and Mann are discussed but not favored particularly because of the increased probability of infection, absorption of fascia, and recurrence. The author seems to feel that the earlier claims made for the fascia lata operations are not being substantiated and, therefore, seems to prefer the use of silk for the deep sutures in these cases. For hernia associated with undescended testicle the Bevan operation is preferred and described.

In discussing recurrence the author states that the general recurrence rate for indirect hernia operations averages 5 to 10% and for direct hernias 10 to 20% (although some surgeons report recurrences as high as 25 to 50%). The presence of an overlooked double sac is mentioned as a frequent cause for recurrence. In discussing mortality, on page 212 the author lists a rate of .83% in 8,000 operations for simple hernia which he collected from the literature, whereas on page 177 he lists a rate of 1% in 6,000 operations collected from fifteen clinics.

The subject of strangulated hernia is well described and illustrated although the arrangement is somewhat confusing in that it is covered in two widely separated sections of the book.

A very excellent treatise on the injection method is given with 69 pages devoted to this subject. This is one of the best works on the injection method available. In agreement with the best practices the author condemns the old caustic solutions and prefers the milder preparations. In his personal series he mentions 94% cured in small indirect hernias, 85% cured in large indirect hernias, and 80% cured in large direct hernias. It will be interesting to note whether he retains his present enthusiasm with the lapse of time.

In discussing femoral hernias he prefers the femoral operation for the non-strangulated type and the more difficult inguinal operation for the strangulated.

Other varieties of hernia are amply described including the umbilical, ventral, diaphragmatic (most of the adult types he believes to be traumatic in origin), internal, lumbar, obturator, sciatic, perineal, and sliding. Hernia of the bladder is also dealt with adequately.

In the medicolegal section the author ascribes much of the confusion as due to the influence of the old English

court decisions that excessive strain must be regarded as an injury. As evidences indicating the existence of hernia prior to an alleged accident the author mentions evidence of truss wearing, relaxed rings, the large size of the hernia, failure of the patient to recall the exact date of onset, the absence of pain at the onset, failure to stop working, neglecting to report the hernia promptly, the presence of a hernia or scar in the other groin, history of pre-existing discomfort in the affected groin, a large and weakened wall on the other side, family history of hernia, and a history of hernia or hydrocele in childhood.

Contrary to the popular notion the author states that the inguinal canal is not weak, stating that, "the semi-sphincteric fibers of the internal oblique exert a lifting power, which is called into play during increased intra-abdominal pressure, and this brings into action a corresponding amount of resistance of the inguinal canal;" hence it is impossible for strain or effort to produce an inguinal hernia unless a patent preformed sac is present. A true traumatic hernia is a great rarity and occurs only when there is a severe external trauma sufficient to cause much disruption and laceration of the tissues, a hernia in such cases contains no sac. The author feels that not more than one in 10 thousand hernias is traumatic.

In discussing the examination of employees before employment the possibility of subsequent hernia developing in individuals with loose rings and relaxed walls is stressed. Individuals with patent but empty, preformed sacs after performing heavy labor for a period of time are likely to have intra-abdominal content enter the sac and thus cause a clinical hernia. This subject is a very important one for it is only through experience and palpating a large number of individuals that one gains the delicacy of touch to ferret out many of these cases of congenital sacs which constitute a subsequent hazard.

The book contains 591 pages with 281 illustrations comprehensively dealing with the subject. An extensive bibliography is listed following most of the chapters. Adequately presenting a subject of constant interest to most physicians it should be a valuable addition to the doctor's library.

Georgia Railway Surgeons

THE SURGICAL ASSOCIATION OF THE ATLANTA AND WEST POINT RAIL ROAD, THE WESTERN RAILWAY OF ALABAMA AND GEORGIA RAILROAD will hold its Nineteenth Annual Meeting at the Atlanta Biltmore Hotel, Atlanta, Georgia, on Tuesday, March 21, 1939, beginning at 8:30 A.M.

The Program includes following presentations:

1. "Osteomyelitis," by DR. H. M. MICHEL, Augusta, Georgia.
2. "Injuries to the Eye," by DR. S. J. LEWIS, Augusta.
3. "Injuries to the Elbow," by DR. R. H. McDONALD, Newman, Georgia.
4. "Roentgenology," DR. R. H. FIKE, Atlanta—this being the President's Address.
5. "Treatment of Fractures in General," by DR. RICHARD BINION, Milledgeville, Georgia.
6. "Injuries to the Kidney," by DR. W. D. McGEARY, Madison, Georgia.
7. "Traumatic Injuries to the Abdomen," by DR. FLOYD W. McRAE, Atlanta.
8. "Shoulder Dislocations," by DR. W. H. CLARK, LaGrange, Georgia.
9. "The Management of Minor Injuries," by DR. JESSE L. WELDON, Lanett, Alabama.
10. "Back Injuries," by DR. EDGAR F. FINCHER.
11. "Coronary Occlusion," by DR. GEORGE A. TAYLOR, Augusta.
12. "Occupational Disease Problems," by C. O. SAPPINGTON, M.D., DR.P.H.

The Officers of the Association for the year 1938-1939 are as follows:

DR. R. H. FIKE, Atlanta, *President.*
DR. P. Y. DONALD, Selma, Alabama, *Vice-Pres.*
MRS. R. E. COOPER, Atlanta, *Sec'y-Treas.*

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Chicago Society

THE CHICAGO SOCIETY OF INDUSTRIAL MEDICINE AND SURGERY held its Mid-Winter Scientific Meeting at Chicago, February 6, 1939. In the absence of DR. EDGAR A. DEGENHARDT who is President of the Society, DR. EMILE C. DUVAL, Vice-President, presided.

The speaker of the evening was DR. PAUL B. MAGNUSON who talked on "Arthritis Complicating Injuries," and illustrated a number of cases.

DR. MAGNUSON's presentation was then discussed by DRS. WILLIAM R. CUBBINS, EDWARD C. HOLMBLAD, JOHN D. ELLIS, FREDERICK W. SLOBE, JOHN G. FROST, FELIX M. JANSEY, and HORACE STIMSON.

From the Archives

AS TO the teeth and occupational diseases, I have seen teeth stained by certain dusts and fumes, particularly copper.* But first let me say I think the question ought to be broadened a little to include the alveolus and the gums and jaw bone; the teeth become carious or, perhaps, are removed because of diseases in the pulp and surrounding tissues. Here the heavy metals especially are prominent: lead, bismuth, thalium, cadmium, phosphorus, and others. Then too, teeth are affected in acid dippings, pickling, etc., from acid fumes. And teeth and gums are subject to a great deal of pathology as a result of exposure to the derivatives of the benzene series, especially the lower benzols, toluol, and their immediate derivatives. And likewise, where exposure has been extensive, to the lower petroleum distillates. Undoubtedly there are others. Teeth are secondarily involved in a considerable number of constitutional conditions, such as those affecting nutrition . . . .

"As to the relationship of cancer and certain occupational diseases, I do not believe, from what we know about the cause of cancer, that occupational diseases are much of a factor in its causation. There are a lot of theories as to the real cause of cancer, but none has been proved, and our pathologists are not agreed. . . .

* From a Round Table discussion of May, 1937.

Industrial Medicine

Reg. U. S. Pat. Off.

The Journal of Traumatic Surgery and Occupational Diseases

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VOLUME 8 MARCH, 1939 NUMBER 3

"The Medical Bulletin"

WHEN "The Medical Bulletin"* came to our desk in its somber brown color, a glance at the cover announcement of the Editorial Board, W. J. DENNO, A. W. SCHOENLEBER, JAMES M. ADAMS, and MCIVER WOODY caused us to spin the pages and prick up our ears. We have known the first two gentlemen for years, and especially DR. SCHOENLEBER when we were medical students and beginning practitioners in Chicago.

"The Medical Bulletin" is published by the Standard Oil Company of New Jersey and intended primarily for distribution to members of its medical staff and its associated companies as a medium of exchange of experience, case reports, and epitome of activities, and to stimulate interest in common problems. It is, however, available to others as long as the supply of a given issue holds out. It was inaugurated in 1932, and appears several times a year, some articles being in foreign languages accompanied by an English translation. The issue before us is chiefly a report of medical statistics and summaries for 1937 by DR. SCHOENLEBER, Medical Director of this Company which operates world-wide, but the later pages carry three most interesting articles, referred to below.

We recall the days when the late DR. W. GILMAN THOMPSON, Professor of Medicine at Cornell University Medical College and Visiting Physician to Bellevue Hospital, was the general medical advisor for this Company, and many will recognize him as the well-known author of the first general text in America on "The Occupational Diseases," published in 1914* — a work which is still standard and often referred to.

THE medical statistics of the present bulletin comprise an international purview, since this Company operates all over the world, and, in fact, its largest group of employees (7213) comprises the Anglo-American of Europe, its third largest the Nederlandsche Koloniale Petroleum Maatschappij (5418), with the Standard Oil of Louisiana in second place (6237). True, if the various subsidiary companies in New Jersey were all combined the figure would outstrip any of these.

The Company, in 1937, employed 32,262 persons in its 15 U. S. affiliated companies, 29,495 in its nine Tropical subsidiaries, and 12,109 in its four European subsidiaries, giving a grand total of 73,866 employees in its 28 affiliates and subsidiaries. Dr. Schoenleber not only analyses these with meticulous care for frequency rates of sickness, injuries, non-effective rates and death rates, with constant comparisons with the preceding year 1936 and occasional comparisons with the past five year experience, but, also (for Tropical countries) 6,705 members of employees' families and 2,577 others.

The whole scheme of analysis, discussion, and deductions is founded on the same basic lines of statistical inquiry so that, for the most part, reliable comparative experiences of the various companies in the three great world groups can be made. Charts and statistical data necessarily accompany, and the reader is profoundly impressed with the vast amount of collected details which have been boiled down to a few pages. As DR. SCHOENLEBER indicates, it is to be regretted that some international scheme of standards is not at hand for use by all operating companies so that other industrial comparisons might be made. Thus, experiences of other great internationally-operating corporations, such as the United Fruit Company, and the rubber and chemical companies might well constitute likely organizations for such comparisons.

The present analysis for 1937 concludes that, while these statistics are accurate, it is still possible that some contained groups report more sickness cases than actually occur as, for example, in outlying plants where dependence has to be placed on more or less non-medical observations. Dismissing the possibility of overstatement, the health record for the Company employees was excellent when compared with other groups for which similar statistics might be considered available.

This point about the possible over-statement of sickness data recalls to our mind the similar finding in Chile, commented upon editorially by IN-

* *The Medical Bulletin*, 30 Rockefeller Plaza, New York. Vol. 3, No. 7: 236-287, November, 1938.

* By D. Appleton & Company.

DUSTRIAL MEDICINE in its November, 1938, issue, where experience showed that 40% of persons seeking treatment were not genuinely sick, or their afflictions were of no importance. On the other hand, medical examination of supposedly well manual laborers revealed over one-fifth with "hidden sicknesses" requiring immediate treatment.

In our issue of December, 1938, we commented editorially on the various state surveys for potential industrial health hazards now being conducted in the United States, four of which were already completed. The present *Bulletin* does not take up the hazards phase, except possibly in a general climatological and racial relationship. It is devoted essentially to a discussion of adult disability, which it classifies under the usual topographical groupings of the body systems—respiratory, digestive, skin, etc., but recognizes a distinct infectious group.

As might be surmised, the respiratory system leads, but with marked differences in zonal relationships. The infectious group is second and with less differences. The digestive system falls third, also with considerable zonal differences. In the United States and Europe, the *common cold* and *influenza* are far and away the leading causes of disability. In the Tropics, *malaria* led, but with the common cold and influenza (both about equal) standing close up.

On the basis of reports received from all 28 companies, health conditions were less favorable in 1937 than the previous year, the increase in frequency rate being equal to 8%. When compared with the last five years, the 1937 rate showed an increase of 24%.

Frequency rates for disabilities (with a general average of 440.13 per 1,000 employed per year, 1937), were highest in the South and Southwestern U. S. (657.62), with England a close second (602.29), and the total for European countries (527.46) surpassing that for the United States (471.82), with the total Tropics considerably less (369.62). The non-effective rate (average daily disabled employees per 1000) was 13.74, which was practically the same as that reported in 1936.

Notable increases were commonly found when the year 1937 was compared with that of 1936 in the various parts of the world for certain leading diseases. For example, for the *common cold*, 63% increase in Europe (where the statistics are more open to question), 42% in Eastern United States, 21% in Central U. S., but a 7% decrease in Southern and Southwestern U. S. A somewhat similar increase was noted for *influenza*, the more loss-time disabling of the two diseases, and sometimes unavoidably inseparable from the common cold.

In Southern U. S., *malaria* proves to be an important industrial problem with a rate of 15 per 1000 employees per year, but much less than that for Tropical Countries, 44.1, except the Canal Zone, where the rate is only 12 per 1000.

Appendicitis in the United States (7.44) leads the world as compared to European countries (4.54) and Tropical countries (3.08). Of peculiar interest is the relatively high rate reported for appendicitis (13.46) in Southern U. S. where it is suspected that improper diagnosis may be a factor. Unfortunately, there are again no data from other groups to confirm the Company's experience that appendicitis is much lower in Eastern than in Southern and Southeastern U. S.

Studies are given for *typhoid* and *para-typhoid* and *amebic* dysentery. We find no mention of *hook-worm disease*, at least statistically considered, which moves us to observe that in Southeastern Kentucky, within the past year, we were informed by a closely observing laboratory worker in a coal mine center that, in something like 2400 employees, the stools of 60% were found to carry hookworm ova, although the disability rate was low from this cause.

THE present *Bulletin* modestly refrains from indicating the size and complexion of the medical organization, finances, hospitals, nursing service, or educational work done by this far-flung industrial organization.

In a general way, disability rates may be said to reflect for mind and body, the effects of community, home, habits, heredity, and work influences, all of which play varying roles in non-effective rates and most of which are subject to further identification by close medical and statistical procedure.

DR. F. L. VASSALLO, in Northern Argentine, submits an interesting report on *bubonic plague* which, for a number of years, has occurred in periodic epidemics at intervals of about two years, coming in the cold, dry season. This has been chiefly of the "bubo" type, but with some septic and pneumonic cases. His report deals especially with the wandering habits of sick rats and the places they seek for shelter, also his conviction that of all rat-riddance instrumentalities, a strong creolin solution, widely used, is the method *par excellence* in conquering this pest. Even the housewife who would catch the ordinary rat should learn that bait should be placed in secluded places difficult of access — even in small cardboard boxes, closed or with only one opening, which is the proper method of enticing this wary rodent to the bait. The present article also gives specimen plates of the pleomorphic forms of *B. Pestis.*

We had always thought that the mosquito, and particularly that specie which is host to the malarial organism, bred in quiet, shaded waterways or in such domestic appurtenances as gutters, rain barrels, and tin cans. However, DR. M. COLQUE RIERA, of Argentine, reports quite the contrary, so again one of our fundamentals is shaken to its base. Here the malarial mosquito (*A. pseudopunctipenis* — the only carrier) lives only in gently flowing streams of pure water, exposed to bright sunlight where algae (*Spirogyra*) grow. Mild shadowing of breeding places is sufficient to kill the algae and provoke the disbanding of the mosquito.

Hard pressed for nurses in outlying environs caused one of the companies in Northern Argen-

tine to create its own school of nursing, reported upon by CECELIA BENNETT and DR. VASSALLO at Manuela Pedraza. The difficulties to be faced to attract young Argentine women with acceptable education and moral background in Latin countries, whose parents are loath to let their young daughters leave home, were finally overcome by constant trading on the moral tone, and high standards set for the school. Unlike the United States, in this district there is a close correlation between education and social standard. Applicants are selected at 16 years of age — this age considered equivalent to 18 in the temperate zone — and required to pass a thoroughly complete physical examination, then placed on a probation period of three months. The young nurses are provided for in every way, including a salary equivalent to $12 per month in the United States. They also have the advantage of a small general hospital where all types of cases may be more closely observed than in a large hospital. Closer contact with supervisors and instructors has enthused both groups while pursuing a standard curriculum such as would be found in the States, at the same time stimulating outside interest. The school was opened September 1, 1937, with eight original students.

When one reviews such a report as this, he is moved to wonder how soon the time will come when all common hazards will be preconceived and scientifically shuttled aside before inevitable disabilities arise. Are we not lingering in the phase of collecting and analyzing data, not that this should be soft-pedaled at all, but that it should be more rapidly appropriated to enormously valuable economic ends in better keeping with progress made in other lines in the past few decades. "This loss happens repeatedly! Why? Has every scientific means been brought to bear to stop it? How soon can it be stopped?"

INDUSTRIAL HEALTH AND DISEASE

What Is Expected of the Physician and Surgeon in Compensation Cases

HAROLD CLARK THOMPSON, LL.B.,
Denver, Colorado

THIS subject involves many matters which are not only in the sphere of medicine but also in the fields of law, administration, and policy.* Consequently, before a detailed discussion of the matters to be presented, it is wise to offer a few introductory remarks concerning the Workmen's Compensation Act of Colorado, its scope and administration.

Briefly, the Workmen's Compensation Act provides for certain medical and compensation benefits to injured employees, and the dependents of employees who have been killed, and requires that the payment of these benefits shall be the responsibility of those employers who are subject to the provisions of the Act.

There are three methods whereby the responsibility of the employer may be fulfilled: (1) by insuring his liability under the Workmen's Compensation Act with a private insurance company licensed to do business in the state of Colorado; (2) by insuring his liability with the State Compensation Insurance Fund; (3) by obtaining a self-insurance permit, which means that the employer by posting certain securities with the Industrial Commission of Colorado, pays his own losses and assumes full responsibility therefor. All public employers, such as the state, counties, schools, towns, and all political subdivisions, are required to carry their compensation with the State Compensation Insurance Fund. The Industrial Commission is charged with the administration of the Workmen's Compensation Act, and it also is the administrative body of the State Compensation Insurance Fund. That is to say, in reality the three Industrial Commissioners are the board of directors of the State Compensation Insurance Fund.

In a quasi-judical capacity they have jurisdiction over all cases and claims, regardless of the method of insurance. Thus the Industrial Commission, acting both directly and through its referees, hears and determines all cases. Their decisions on the facts presented in each case are final and are not subject to review by the courts except on such matters as involve questions of law.

Since there are three methods of insuring the payment of compensation benefits, and since the State Compensation Insurance Fund, which in reality is a state operated mutual insurance company, is the department with which I am connected, I will speak on matters of policy from the standpoint of the Fund. As it is both possible and probable that some of the private insurance companies and the self-insurers may have other or better methods than those used by the State Compensation Insurance Fund, what I have to say is chiefly the result of observations during my years of association with the Fund.

* Read before the Sixty-eighth Annual Session of the Colorado State Medical Society, Estes Park, Colorado, September 9, 1938; published in *Rocky Mountain M.J.*, December, 1938.

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SUBJECT	GRUEL MEAL	GRUEL MEAL PLUS FOUR ASPIRIN TABLETS	GRUEL MEAL PLUS FOUR ALKA-SELTZER TABLETS
	MINUTES	*MINUTES*	*MINUTES*
E.B.	90	90	60
V.B.	90	120	75
C.K.	75	120	75
E.P.	75	90	90
T.C.	105	150	90
M.C.	90	135	75
AVERAGE	88	118	78

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2. **The average emptying time of the stomach after consumption of the test meal followed by aspirin was 34 per cent greater than the average time for the meal alone, and 51 per cent greater than the average time for the meal followed by Alka-Seltzer.**

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I shall discuss those things which deal with the routine and technic of handling compensation cases, insofar as the State Compensation Insurance Fund is concerned. When an employee is injured in an industrial accident he is required by law to report that accident to his employer within two days, and his employer is required by law to file a report of accident on forms prescribed by the Industrial Commission within 10 days from the date of the accident. Penalties are provided in the statute for the failure of either the employee or the employer to carry out the mandate of the law.

The report of accident from the employer, or a claim from an injured employee, is the foundation of the file in the particular case in both the offices of the Industrial Commission and the State Compensation Insurance Fund. Doctors sometimes wonder why, when they send in reports and bills, payment is not immediately forthcoming. The reason is that we do not have a legal file until a report of the employer or, if the employer refuses to file a report, the claim of the injured employee, is properly filed and docketed.

In addition to the report of the employer it must follow as a matter of routine that the report of the attending physician must be received by the insurance carried before action on the claim can be taken. Many members of the medical profession have inquired at various times regarding the reason why the insurance carrier continually writes for reports, and the reason for the so-called red tape. The information called for in these reports is vitally necessary to the claim department of any insurance carrier in the adjusting of the claim.

The computation of compensation benefits is almost purely a mathematical problem, and since mathematics is an exact science, obviously, exact information with respect to date of accident, nature of injury, and the probable length of disability is necessary in any procedure whereby an attempt is made to ascertain the compensation benefits.

The importance of making reports promptly cannot be over-emphasized. You are all familiar with the saying that an army moves on its stomach. With us, the moving of a claim through the processes of adjustment depends upon information, particularly medical information. It can be said that to us a doctor is just as good as his reports. A man may be one of the finest surgeons in the country. He may have performed a perfect operation, or obtained a splendid result in a bad fracture, but unless he goes further and supplies us with the required reports, supplementing them by letter where necessary concerning the history of the accident, the diagnosis made, and the estimated probable period of disability, he has failed as an industrial surgeon. He will cause himself delay in the payment of bills, the writing of letters, and possible loss of time in appearing at hearings. Further, if too dilatory, he will eventually incur the ill will of his patient, because in no case is compensation paid to the claimant unless medical information which justifies it is in the file of the insurance carrier.

While I have stated that action is not commenced on a case until a report of the employer is received in the office of the State Compensation Insurance Fund, it is true that in a few cases the life or health of the patient will not warrant delay incident to the completion of routine matters. Often emergency operations or similar procedure must be undertaken. What should the physician or surgeon do in such cases?

The answer has to be a practical one. Every physician or surgeon in his general practice is sometimes called upon to take such procedure for the purpose of saving a life, without taking thought of his remuneration or the source from which it is coming. He should do the same thing in compensation cases. It is true that in some such instances it will perhaps later be found that the case is not covered under the Compensation Act, but I believe that a survey of such instances would show no higher percentage in compensation cases than

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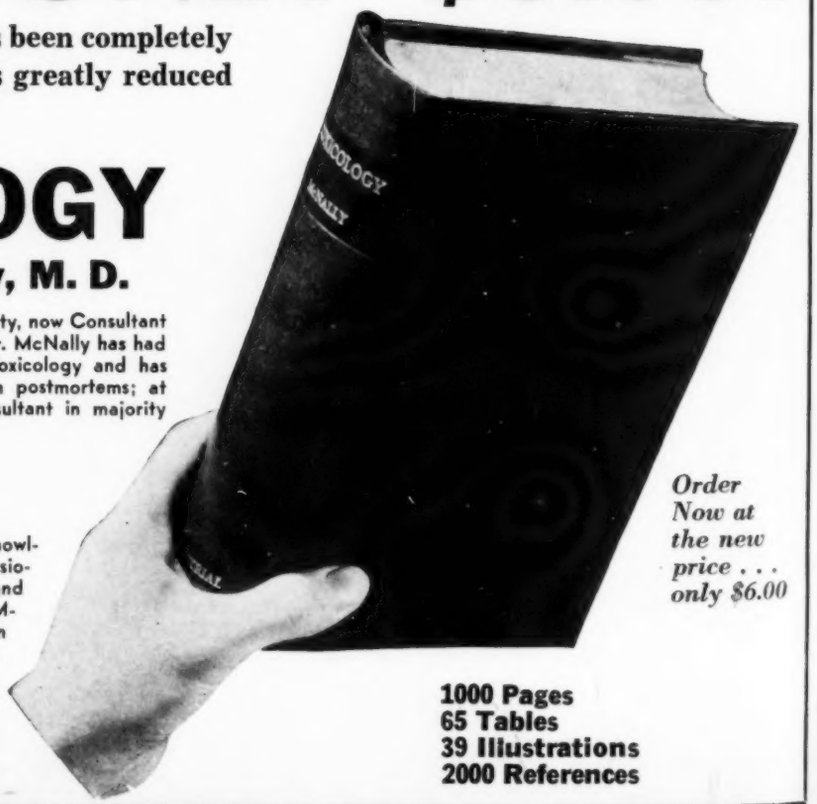

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in those cases taken in private practice under similar emergency circumstances.

Then too, as a practical solution in extraordinary cases, it is our suggestion that the attending physician or surgeon call our office by telephone. This is the procedure followed in Denver and it is often possible for us to obtain enough facts by telephone to justify us in accepting liability even before the report of the employer is actually filed. For physicians and surgeons who reside throughout the state, we suggest the long distance telephone. It is not the desire of the office by this suggestion to encourage a flood of long distance telephone calls, but in any case where the emergency is grave and immediate action must be taken, and where the physician feels that some authority should be received from us, we welcome such procedure.

In considering operations, the subject should not be passed without discussing herniotomy. The Workmen's Compensation Act of Colorado makes a distinction between hernia cases and cases involving ordinary trauma. In order to be compensable a man who has incurred a hernia must prove two things: (1) that the appearance of the hernia was accompanied by pain; (2) that it was immediately preceded by some accidental strain suffered in the course of the employment. In other words, the fact that an accidental strain may cause a pre-existing hernia to become larger or to strangulate, does not make the case compensable. Due to this fact, and to the requirements of the statute, hernia cases are always carefully scrutinized. Attending physicians should not, therefore, operate in hernia cases where delay of the operation does not endanger the claimant's life. They should receive consent of the insurance carrier before performing the operation, because liability is not accepted in some hernia cases since the carrier may have information indicating the man's hernia existed prior to the alleged accidental strain. If the situation is an emergency, and there is time, the surgeon should call our office explaining the facts, and he will be advised whether the office will accept liability on the strength of his phone call or whether further investigation may be had.

Many surgeons throughout the state phone us in serious cases and discuss the matter thoroughly. We welcome such procedure, as it enables us in many cases to accept liability at once, or in doubtful cases to warn the physician, that he may protect his own rights.

X-Rays

THE problem of x-rays is a difficult one. Naturally it is our desire that a sufficient number of x-rays be taken to diagnose the case properly. It is not the desire of the Fund to have x-ray bills beyond reason, nor to have x-rays taken for which there is no actual need. Conversely, it is not the desire of the Fund to have the physician fail to take necessary x-rays only for saving expenses.

In the course of our experience we have found cases where the doctor, in our opinion, has taken an unnecessarily large number of x-rays. On the other hand, we have had cases where the attending surgeon, by not taking more x-rays, has overlooked important injuries and disabilities. For example, we have had a few cases of fractures of the os calces and x-rays of the feet were made. Later on, a more thorough examination and x-rays of the spine revealed compression fractures for which treatment should have been rendered at the time of the accident.

The happy medium is, of course, the object which we seek to attain—namely, sufficient x-rays properly to diagnose the case, and the elimination of unnecessary plates which do not add to information concerning the claimant's disabilities.

One matter in industrial cases which needs emphasis is that of the confidential relationship between doctor and patient. The compensation law practically abolishes this relationship so far as matters relat-

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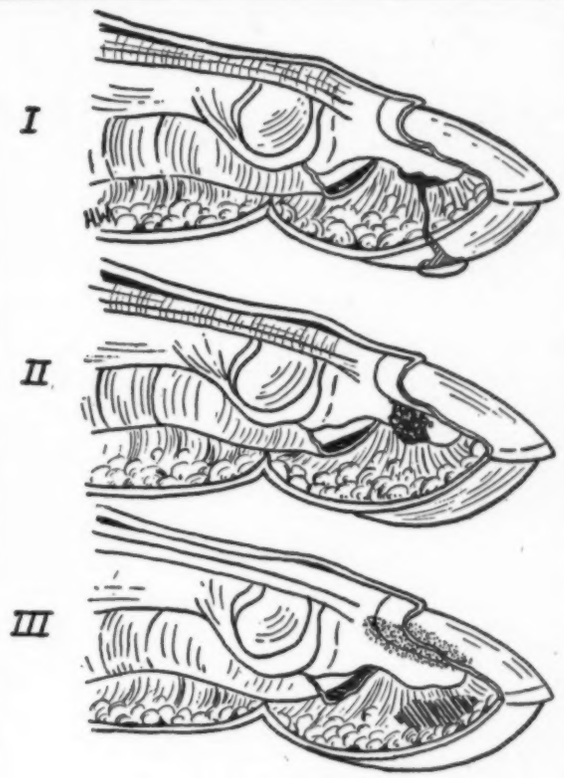

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ing to industrial accidents are concerned. Section 81 of the Workmen's Compensation Act provides that any physician who has attended an employee in a professional capacity may be required to testify before the Industrial Commission when it so directs, although a physician is not required to disclose confidential matters imparted to him which are not necessary to a proper understanding of the case. Thus "the proper understanding of the case" is capable of broad interpretation. Where a history of venereal disease, for example, is a vital element to the proper understanding of the case, any physician who has attended the man for such a disease may be required, and often is, to impart full information concerning the matter to the Industrial Commission. The purpose of this section of the law is in keeping with the entire purpose of the Act, namely, to try as far as possible to arrive at the truth of any given situation, i.e., the true causes of disability and proper methods of alleviating it.

Consequently, where the physician possesses information from prior treatment of the injured claimant which would influence the decision as to whether the case is compensable or as to the extent of temporary or permanent disability, it is his duty to inform the insurance carrier. If he is doubtful of the advisability of imparting said information or fears legal action for breach of confidential communications he should inform the insurance carrier that he is in possession of information bearing on the case and that he is doubtful as to his right to impart it. The insurance carrier is then warned, and can obtain from the Industrial Commission an order requiring the claimant to consent to the disclosure of the information involved. In this way the rights of all parties are fully protected.

Focal Infections

ONE of the most disturbing factors in the handling of compensation cases is the part played in injuries by pre-existing pathological conditions or focal infections. With one or two exceptions the law as now interpreted holds that where an injury aggravates a pre-existing disease the insurance carrier is liable for the disability resulting from the combination of the two — for example, cases of fractures or wounds which will not heal because of pre-existing lues. It ultimately, then, becomes our burden to treat the injury and also to eradicate the systemic disease. Similarly, in back or joint cases, pre-existing foci of infection may prevent proper healing. To accomplish the desired result and restore the man to industrial usefulness, it becomes our task to remove the various foci, and we have tonsillectomies, dental work, and other things done, as required.

One exception, perhaps, in the law as now interpreted, is that of hernia cases. Even though an accident may enlarge a pre-existing hernia and thus aggravate the hernia, such aggravation is still not compensable due to the special section of the law relating to herniae.

The word "aggravation" is a broad one. What the Fund expects, and what, I am sure, any insurance carrier expects of the attending physisian or surgeon in such cases, is fairness. If the accident actually did aggravate some pre-existing condition, we should be promptly advised, and we are usually glad to assume the burden the law imposes upon us. On the other hand, we do not believe that groundless speculations should be indulged in nor tenuous theories advanced merely for the purpose of making a case compensable.

Estimation of Permanent Disability

BEFORE discussing methods of estimating permanent disability, it is well to mention the different types of disability from a compensation standpoint. In any case there may be as many as four periods: (1) temporary total disability, i.e., the period during which the patient is under treatment and is totally disabled from any work: (2) temporary

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partial disability, the period during which he is well on the road to recovery, may or may not be still under treatment, is still unable to return to full duty, but may be able to do some light work: (3) permanent partial disability, i.e., that disability which remains in the body as a whole, or in some member thereof after treatment has ceased and maximum improvement has been reached; and (4) permanent total disability. The latter term, of course, is self-explanatory and applies to those cases where the man remains permanently and totally disabled following his accidental injury.

The estimation of permanent disability percentages is a difficult phase of industrial surgery and one which requires both experience and skill. Because disability questions invade the realms not only of medicine and law, but those of economics, sociology and others as well, the ability to measure trauma accurately as an industrial handicap is becoming increasingly regarded as a specialty. We shall turn first to some legal considerations.

The Colorado Workmen's Compensation Act provides for two methods of estimating permanent disabilities, i.e., the table of specific losses, whereby the man is allowed a certain loss or partial loss for the various members of the body, such as the loss or partial loss of a hand or an eye; and the other method whereby disability is measured on the body as a whole as a working unit. Normally, where the injury is confined to one member, the specific schedule alone is used. Injuries in which the working unit basis is followed are those where more than one member of the body is affected, or those which by their nature impair the function of the entire body. Since there is sometimes a wide variation between the estimates given by different examiners, the crying need in compensation work is some method of standardization.

With this problem in mind, some eminent members of your profession have already undertaken serious and scientific study of the question. Kessler of New Jersey has done some fine work, and his book contains many valuable suggestions. McBride of Oklahoma embarked upon a still more ambitious program by trying to reduce the measurement of permanent disability to the nicety of a mathematical formula; and, while some of his recommendations probably could be carried out only in a highly equipped institution, still his theories and suggested standards offer much to the surgeon who is trying to evaluate a given condition according to the rules of fairness and justice.

In spite of all well-intentioned efforts to reduce the problem to one of cold science, however, I suppose there will always be some differences

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of opinion, especially in those borderline cases where trauma merges into pathologic conditions or disease. Until science catches up with the problems presented, we shall all have to do the best we can. Then, too, doctors are a cross-section of humanity, just as any other group. Some of them are supersensitive, highly susceptible to pain, and extremely sympathetic. Others may be of sterner fiber. We expect the estimates of the former to be higher than the normal average, and those of the latter to be lower. Both groups may be intellectually honest and yet inaccurate in their opinions. What we most desire is the estimate of the man who can be liberal and fair, can look at the patient he is examining objectively, and who has had the education and experience necessary to give us as nearly as possible the exact truth of the case. Our own position is that if a claimant has a certain disability, he should be paid for it; and if he does not have it, he should not.

No paper on the subject at hand would be complete without brief mention of a few matters of policy, brief consideration of limits of liability, and reference to a few cardinal principles.

First, it must at all times, be remembered, as McBride has said, that three persons or groups are vitally interested in an industrial case: (1) the injured employee; (2) the employer or insurance carrier; and (3) the Industrial Commission. The first must receive the care and attention which the injury demands. The last two must know how and when the man was injured, how long he will be disabled, and whether he will have any permanent disability.

Second, accurate and detailed records and histories must be kept in all cases both by the physician and the administrative bodies, because the rights of all parties in interest are vitally affected thereby. Since, under our Colorado law, a case may be reopened by the Industrial Commission at any time in its discretion, important questions may arise years after the original injury. Their solution should not be made to depend upon memory or incomplete records. Much theoretical testimony could be obviated and many injustices both to employer and employee eliminated if the original facts surrounding the accident had been carefully and intelligently preserved.

Third, under the Colorado law medical liability is limited to $500.00, and to four months from the date of the accident. This limit can be exceeded by the insurance carrier where by so doing permanent disability can be reduced. Where the question of exceeding the limit comes up, however, the physician should always consult with the insurance carrier in advance.

Fourth, any injured employee should receive the best medical treatment available and in those serious cases where specialized treatment is indicated, it should be given by those specially skilled in that particular phase of the case.

Fifth, in formulating medical opinion, objective symptoms are still the best evidence; that subjective complaints are at best but secondary evidence, except in those cases where confirmed by objective findings or where by the very nature of the case the subjective complaint becomes an objective sign in itself regardless of its truth or falsity.

Sixth, in those many cases where controversy has arisen and hearings are held, no doctor, be he the witness for the insurance carrier or for the claimant, should advance theories or voice opinions which he would hesitate to present in a paper to this society where he knows that his views are being scrutinized by fellows whose education and training are such as to enable them to judge of his sincerity and ability.

Seventh, and lastly, we, as an organization of laymen, wish to stand on this proposition: that all we desire to do in any case is to arrive at the absolute truth, unvarnished and unadorned. The goal thus sought may be difficult, and in some instances impossible of achievement because of our limited knowledge and because medicine itself is still an inexact science. In such cases it is our honest desire to be placed in possession of those facts and to be led along those paths of medical opinion which will most nearly approach our objective.

(Continued from page 1)

DR. HAYHURST says, with reference to his translation of CONROZIER and MAGNIN, on "Silicosis—Case Reports" (p. 114): "I think these authors have hit upon the best definite proof that we have yet had of the autonomous character of silicosis (as distinct from tuberculosis as a factor)." . . . MENTION of these articles does not mean a preference over the others in this issue. Each of the articles carries its own special excellence and timely significance. Each represents the author's BEST. . . . THE editorial (p. 122) comments on "The Medical Bulletin" published by Standard Oil of New Jersey as an expression of its Medical Department. The comment is by DR. HAYHURST. . . . THE service indicated by the postcard, on the insert just ahead of Page 1 is our own expression of a desire to make the pages of this Journal as useful to both readers and advertisers as complete acquaintance can make them. We welcome the cards as they come. And we reaffirm our aim to let no request fall short of the fullest possible answer. This applies even to matters and things of interest to readers which may not be listed on the insert. Thus the card may be used for extra copies, as they are needed; for reprint requests; for specific information on a problem, or a reference; or even for Book Orders!

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